PB90-104795

Superfund Record of Decision (EPA Region 4): Brown Wood Preserving Site, Live Oak, Florida (First Remedial Action) August 1988

(U.S.) Environmental Protection Agency, Washington, DC

8 Apr 88

U.S. Department of Commerce
National Technical Information Service
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Brown Wood Preserving, FL
First Remedial Action - Final

5. Report Date 04/08/88

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Washington, D.C. 20460

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15. Supplementary Notes

16. Abstract (Limit: 200 words)

The 55-acre Brown Wood Preserving site is located approximately two miles west of the City of Live Oak, Suwanee County, Florida. The site is located in karst terrain in which sinkholes are a common geological feature. The areas surrounding the site are considered rural and light agricultural. There are four private wells located along the site periphery that obtain water from an aquifer 20-100 feet below the site. The public water supply wells for the City of Live Oak are located less than two miles away. The site contains a former wood preserving plant facility, which pressure treated timber products with creosote and some pentachlorophenol (PCP) for thirty years between 1948 and 1978. During this time, several different companies operated the facility. In addition, the facility was rebuilt following a fire in February 1974. Sludge and contaminated soils have been identified in the immediate vicinity of the plant site and an upgradient lagoon. This three-acre lagoon drains approximately 74-acres and contains water provided above approximately 3,000 yd^3 of creosote sludge and contaminated soil. In addition, small amounts of solidified creosote and PCP are contained in onsite storage tanks and retorts. In 1981, EPA was notified by one of the former facility owners that hazardous waste may have been handled at the site. In July 1982, the Florida Department of Environmental Regulation (FDER) inspected the site and detected a
(See Attached Sheet)

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17. Document Analysis a. Descriptors
Record of Decision
Brown Wood Preserving, FL
First Remedial Action - Final
Contaminated Media: sediments, sludge, soil, sw
Key Contaminants: organics (creosote, PAHs)

b. Identifiers/Open-Ended Terms

c. COSATI Field/Group

Availability Statement

19. Security Class (This Report) None

20. Security Class (This Page) None

21. No. of Pages 366

22. Price

(See ANSI-Z39.18)

See Instructions on Reverse

OPTIONAL FORM 272 (4-77)
(Formerly NTIS-35)
Department of Commerce

EPA/ROD/R04-88/034
Brown Wood Preserving, FL
First Remedial Action - Final

16. ABSTRACT (continued)

number of organic compounds. A action, completed in February 1988, resulted in the removal of approximately 200,000 gallons of lagoon water and 15,000 tons of contaminated lagoon sludge and soil. The primary contaminants of concern affecting the soil, sediments, sludge, and waste water are creosote constituents including PAHs.

The selected remedial action for this site includes: removal and treatment, if necessary, of lagoon water with discharge to a POTW; excavation, treatment, and offsite disposal of approximately 1,500 tons of the most severely contaminated soil and sludge; onsite biodegradation of approximately 10,000 tons of the remaining soils in a 14-acre treatment area constructed with a liner and an internal drainage and spray irrigation system; covering of the treatment area with clean fill after bioremediation; and ground water monitoring. The estimated present worth cost for this remedial action is $2,740,000.

j.a

United States
Environmental Protection
Agency

Office of
Emergency and
Remedial Response

EPA/ROD/R04-88/034
April 1988

PB90-104795

EPA

Superfund Record of Decision:

Brown Wood Preserving, FL

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DIRECT QUESTIONS RESULTING FROM ILLEGIBILITY TO:

U.S. EPA
OSWER - HSCD

Record of Decision

for

the

Brown Wood Preserving Site

Live Oak, Florida

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ACRONYMS

AMAX/Brown	AMAX Environmental Services, Inc. and the James Graham Brown Foundation, Inc.
CERCLA/SARA	The amended Superfund Law
EPA or the Agency	U.S. Environmental Protection Agency
FDER or the State	Florida Department of Environmental Regulation
LDR	RCRA Land Disposal Restriction Regulations ("Land Ban")
NCP	The National Contingency Plan
ORD	Office of Research and Development
OSWER	Office of Solid Waste and Emergency Response
OTA	Office of Technology Assessment

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RECORD OF DECISION

REMEDIAL ALTERNATIVE SELECTION

I. DECLARATION

A. SITE NAME AND LOCATION

The Brown Wood Preserving Site is located at the intersection of Sawmill Road and Goldkist Road, west of the City of Live Oak, Suwannee County, Florida. The Site is situated in the northwest quarter of Section 22, Township 2 South, Range 13 East.

B. STATEMENT OF BASIS AND PURPOSE

This decision document represents the selected remedial action for this Site developed in accordance with the Comprehensive Environmental Response, Compensation and Liability Act of 1980 (SARA), and to the extent practicable, the National Contingency Plan (40 CFR, Part 300), (NCP). I have determined that the below-mentioned description of the Selected Remedy for the Brown Wood Preserving Site is a cost-effective remedy and provides adequate protection of the public health, welfare, and the environment. These activities will be considered part of the approved action and eligible for Trust Fund monies.

The State of Florida has been consulted and agrees with the approved remedy.

This Record-of-Decision is based upon the administrative record which describes site-specific conditions and the analysis of effectiveness and cost of the remedial alternatives for the Brown Wood Preserving Site. The attached index identifies the items which comprise the administrative record upon which the selection of the remedial action is based.

C. DESCRIPTION OF THE SELECTED REMEDY

- Removal of selected contaminated materials to an EPA-approved hazardous waste facility.

- Onsite biodegradation of remaining, less severely contaminated soils in a treatment area that is constructed with a liner and an internal drainage and spray irrigation system. After the bioremediation is complete the land treatment area will be covered with clean fill.

- Operation and maintenance (O&M) activities will include:
  - Semi-annual groundwater monitoring for five (5) years (See Section 121(c), CERCLA/SARA.) after the completion of the construction activities.
  - Activities necessary to the proper functioning of the bioremediation process and the attendant systems.
  - Additional O&M activities may be identified during the remedial design.

D. DECLARATIONS OF CONSISTENCY WITH CERCLA AS AMENDED BY SARA AND THE NCP

The selected remedy is protective of human health and the environment attains federal and state requirements that are applicable or relevant and appropriate, (or that a waiver can be justified for whatever ARARs which will not be met), and is cost-effective. This remedy satisfies the preference for treatment that reduces toxicity, mobility, or volume as a principal element. Finally, it is determined that this remedy utilizes permanent solutions and alternative treatment (or resource recovery) technologies to the maximum extent practicable.

I have also determined that the action being taken is appropriate wh balanced against the availability of Trust Fund monies for use at oth sites. In addition, the selected remedy is more cost-effective than other remedial actions, and is necessary to protect the public health, welfare or the environment. All off-site disposal shall be in compliance with the existing policies of EPA.

If additional remedial actions are determined to be necessary, a Record-of-Decision will be prepared for approval of the future remedial action.

APR 0 8 1988

Date

Greer C. Tidwell
Regional Administrator

II. DECISION SUMMARY

A. SITE NAME, LOCATION AND DESCRIPTION

The Brown Wood Preserving Site is located at the intersection of Sawmill Road and Gold Kist Road, approximately two (2) miles west of the City of Live Oak, Suwannee County, Florida. The Site is situated in the northwest quarter of Section 22, Township 2 South, Range 13 East (See Maps 1,2, and 3). The 55-acre Site is located in karst terrain in which sinkholes are a common geological feature. The Site is not located in a floodplain; the topography on-site varies in elevation from 85 feet above mean sea level (amsl) to 111 amsl. The areas surrounding the plant site is considered rural and light agricultural. A sawmill and a construction company are located to the east and west, respectively, of the Site. There are four (4) private wells located along the periphery of the Site. The public water supply wells for the City of Live Oak are located less than two (2) miles away. Domestic water in the vicinity of the Site is produced by means of wells into the Floridan Aquifer.

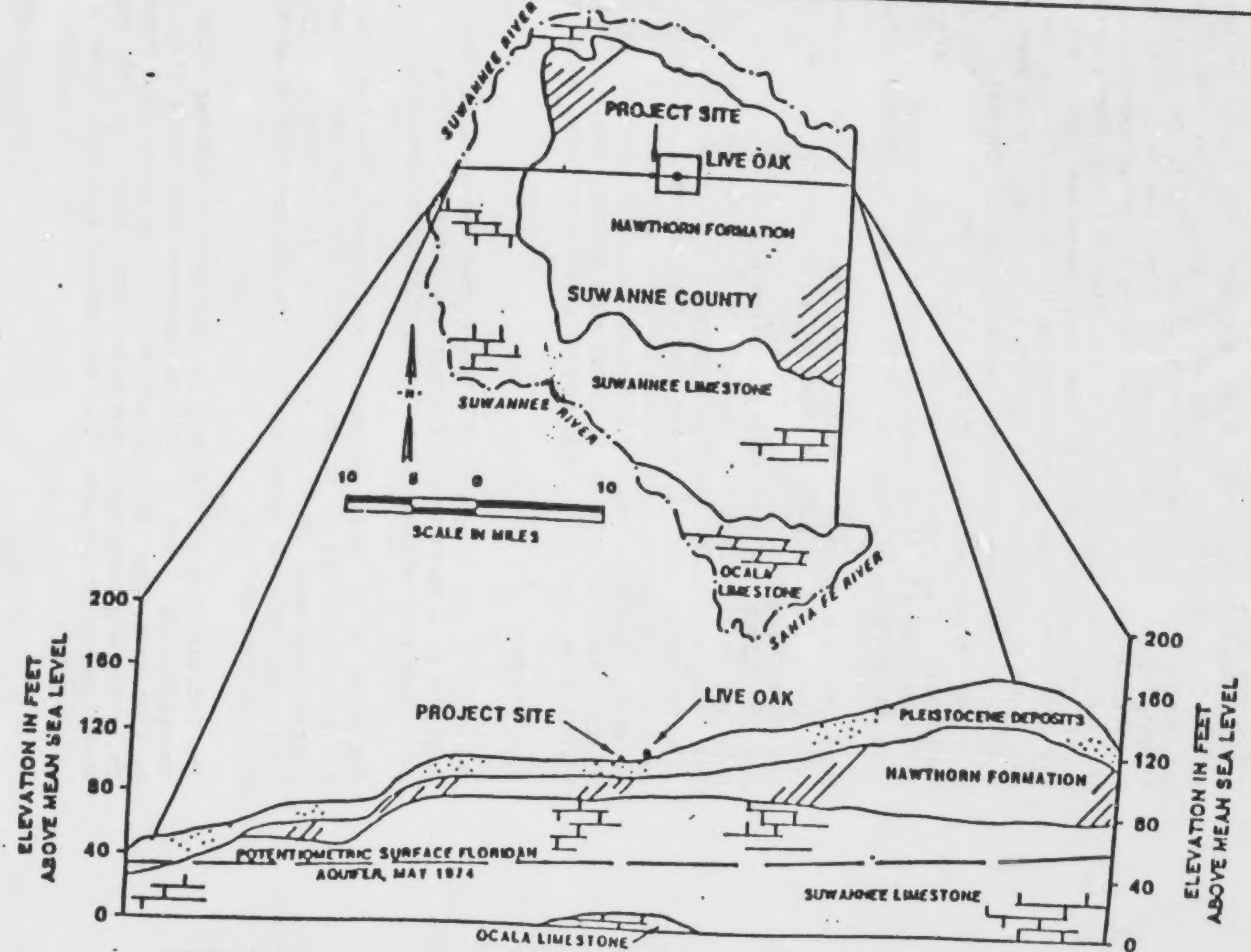
The Site is underlain by karstic terrain consisting of limestone and dolomite. This bedrock is reported to be over 2,500 feet thick with the more permeable deposits contained within the upper 400 feet. The Suwannee Limestone lies from approximately 20 to 100 feet below the Site and is the uppermost unit of the Floridan Aquifer. Portions of the Floridan Aquifer are honey-combed with dissolution chambers and solution pores that allow for the rapid movement of groundwater. A peanut-shaped three-acre lagoon draining approximately 74-acres presently contains about 3,000 cubic yards of creosote sediments/ sludge. Several sets of railroad spur tracks, assorted building foundations, and a few old, rusted steel structures exist on-site.

B. SITE HISTORY

The Site is the location of a plant that was used to treat lumber products by a number of different operators. Portions of the Site have been and continue to be under different ownership.

The plant is no longer in operation; however, the facility was used for pressure treating timber products with creosote and some pentachlorophenol (PCP) for thirty (30) years from 1948 through 1978. During this time, the facility was operated by several different companies.

The facility was constructed in 1948 by W.F. Bancker & Son and operated until March 1, 1952 as Suwannee Wood Preserving, Inc. At that time, the facility was sold to the Brown Wood Preserving Co., Inc. which operated the plant until 1965. In 1965 the Brown Wood Preserving Company, Inc. was merged into W.P. Brown & Sons


SUWANNEE RIVER
PROJECT SITE
LIVE OAK
HAWTHORN FORMATION
SUWANNE COUNTY
SUWANNEE LIMESTONE
SUWANNEE RIVER
N
10 5 0 10
SCALE IN MILES
OCALA LIMESTONE
SANTA FE RIVER
200
160
120
80
40
0
ELEVATION IN FEET ABOVE MEAN SEA LEVEL
PROJECT SITE
LIVE OAK
PLEISTOCENE DEPOSITS
HAWTHORN FORMATION
POTENTIOMETRIC SURFACE FLORIDAN AQUIFER, MAY 1974
SUWANNEE LIMESTONE
OCALA LIMESTONE
VEENSTRA• 75760

GEOLOGIC MAP AND GENERALIZED EAST-WEST HYDROGEOLOGIC CROSS SECTION FOR SUWANNEE COUNTY, FLORIDA

Figure 1.0

Lumber Company, Inc. which operated the plant until 1971. In August 1971, the lumber company was dissolved and the assets taken over by the James Graham Brown Foundation, Inc. which operated the Plant until the facility was closed due to a fire on February 7, 1973. The property was purchased on April 2, 1974 by Mr. W.F. Belote who rebuilt the plant and ran a small number of charges through one pressure cylinder.

On December 24, 1976, Amax Forest Products, Inc. purchased the plant and operated it intermittently until June 22, 1978 when it was bought by Live Oak Timber Treaters, Inc. who may have operated the facility for a short time thereafter. The facility is currently owned by Mr. J. Paul Crews.

The facility is a wood preserving plant consisting of two horizontal retorts (cylinders), a series of storage tanks, and a boiler and associated storage yards and a wastewater lagoon. The present facility layout is shown on Figure 3.0 and described below. The plant layout prior to the fire in 1973 is not definitely known and may have varied somewhat from the existing facility layout.

Creosote was stored in two (2) 50,000 gallon storage tanks immediately east of the retorts. Located east of the southern creosote tank is a 50,000 gallon tank used for storage of pentachlorophenol (PCP). North of the PCP tank is a mixing tank and a 100,000 gallon tank used for petroleum storage. PCP was received at the Site in solid form, mixed with petroleum in the mixing tank and then stored in the PCP storage tank.

Between the petroleum storage tank and the PCP tank is a water storage tank. Process water was obtained from a well located northwest of the water storage tank. North of the creosote storage tank is a concrete slab on which pumps are located. At the south end of the retorts are an oil/water separator and an evaporator.

Steam used to heat the creosote was generated in the boiler located on the east side of the petroleum storage tank. The boiler was fueled by wood chips and fuel oil.

Operations at the plant are believed to have occurred as follows. Untreated timbers were received by rail and possibly by truck, then stored in the area north of the plant until treated. The untreated timbers were loaded onto small rail cars and moved into the retorts for treatment. Treated timbers were removed from the retorts and allowed to dry along a track area immediately north of the retorts. After drying treated timbers were moved to the timber storage area north of the treatment plant. Timbers were shipped to buyers by rail and truck from the storage area. The majority of products produced at this Site were treated with creosote; however, at some time during the plan operation, PCP was also used.

Plant wastewater was discharged to the oil/water separator and from there to the lagoon via a culvert and drainage ditch. Creosote recovered from the oil/water separator was discharged to the evaporator located south of the separator where it was heated by steam to evaporate any remaining water. The creosote was then returned to the storage tanks, or if unusable, it was pumped to the off-specification storage tank located north of the plant.

Sludge and contaminated soils have been identified in the immediate vicinity of the plant site and the lagoon. Water is ponded in the lagoon above sludge and contaminated soils. In addition, small amounts of solidified creosote and PCP are contained in storage tanks and retorts remaining on the Site.

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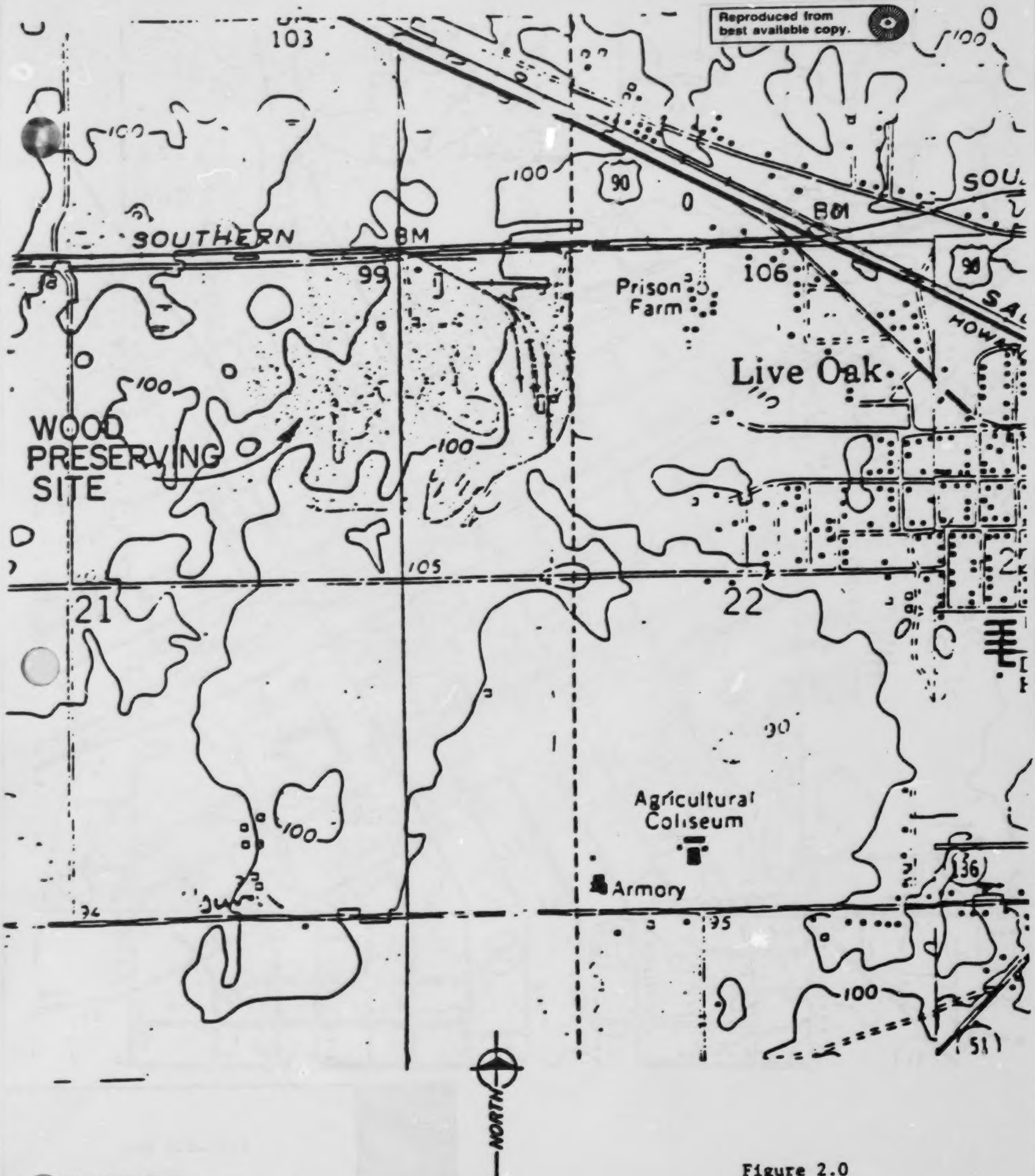
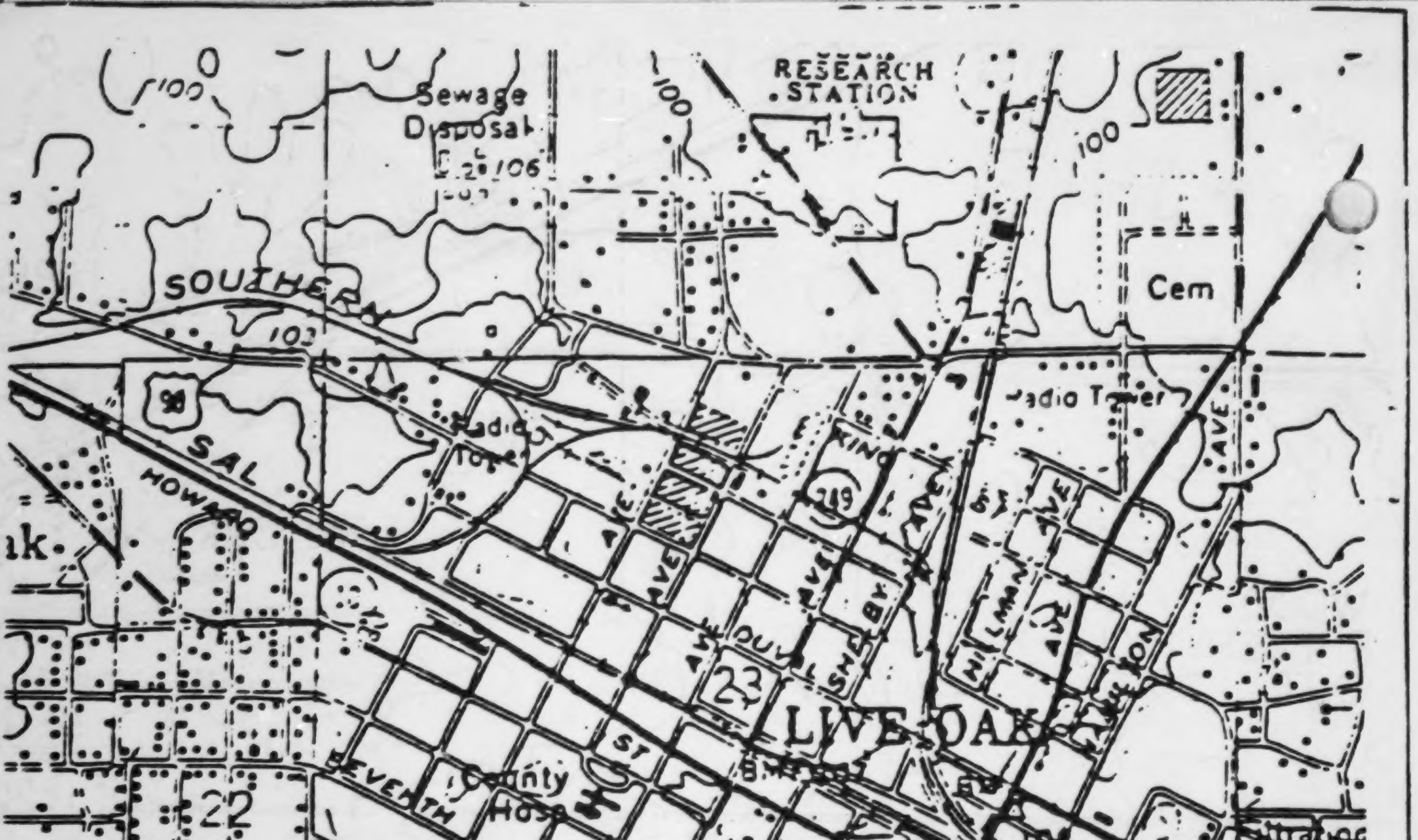

103
100
SOUTHERN
BM
99
90
0
BM
106
SOU
SA
HOWA
Prison Farm
Live Oak
WOOD PRESERVING SITE
100
105
21
22
90
Agricultural Coliseum
Armory
95
36
100
51
NORTH

Figure 2.0

MAPS PREPARED BY
EOLOGIC SURVEY
, 1961

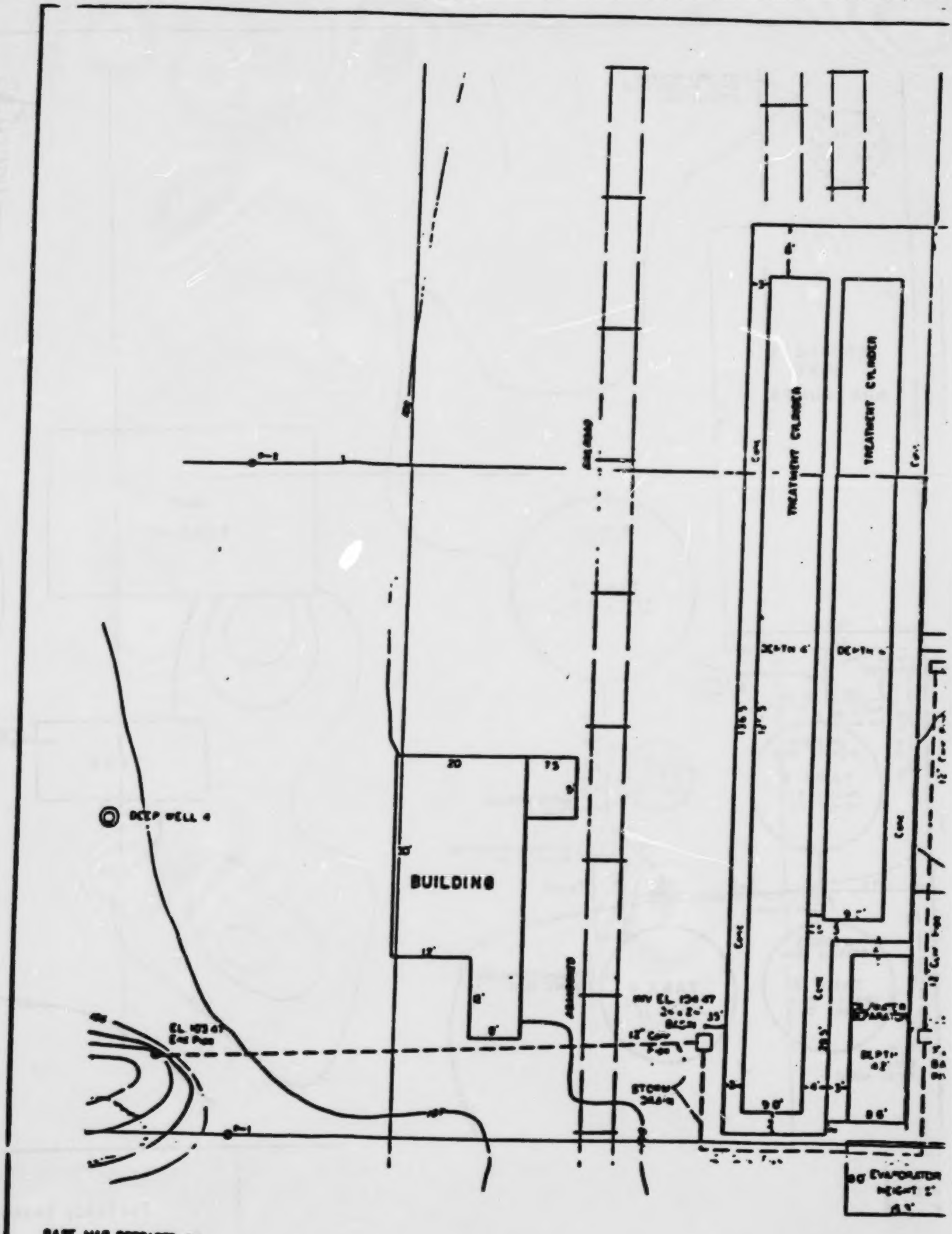
SCALE: 1" = 1000'


RESEARCH STATION
Sewage Disposal
106
100
SOUTHERN
102
Cem
90
SAL
HOWARD
Radio Tower
Radio Tower
KING
129
AVE
DUVAL AVE
SHELBY AVE
AVE
23
LIVE OAK
22
SEVENTH ST
County Hosp
ST
Reeves Field
County High Sch
Court House
Douglas High Sch
BM
Post Office
Radio Tower
Filtration Plant
ST
WALKER
ORVIN
SCRIVEN
HOUSTON
MAPLE
ST
OHIO
Lake Mary
HELVESTON
136
ELEVENTH ST
102
CHURCH
136
PINE
249
100
10
ST
AVE
51

Vicinity Map

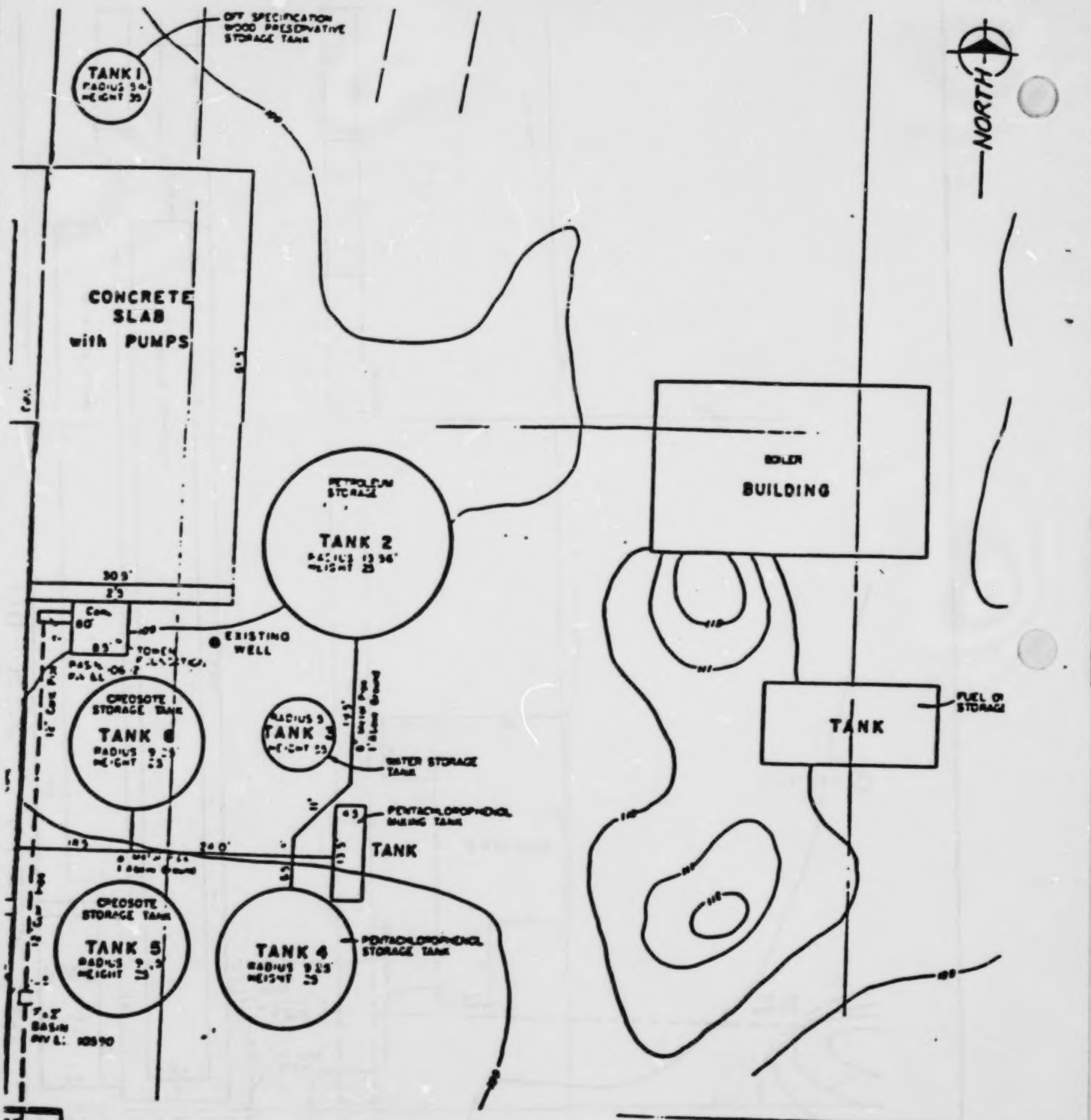
Figure 2.0

RETEC


TREATMENT CYLINDER
TREATMENT CYLINDER
DEPTH 6'
BUILDING
20
DEEP WELL 4
EL. 107.47
INV EL. 106.47
STORM DRAIN
12" CMP
EVAPORATOR
HEIGHT 2'
DEPTH 4.2'
9.0'
9.6'
6'

BASE MAP PREPARED BY
LOWE ENGINEER INC.

Figure 3.0



OFF SPECIFICATION WOOD PRESERVATIVE STORAGE TANK
TANK 1
RADIUS 5.6
HEIGHT 35
NORTH
CONCRETE SLAB with PUMPS
PETROLEUM STORAGE
TANK 2
RADIUS 13.96'
HEIGHT 23
BOILER
BUILDING
30.5'
EXISTING WELL
CREOSOTE 1 STORAGE TANK
TANK 6
RADIUS 9.25'
HEIGHT 23
RADIUS 5
TANK 3
HEIGHT 25
WATER STORAGE TANK
FUEL OIL STORAGE
TANK
PENTACHLOROPHENOL MIXING TANK
TANK
24.0'
CREOSOTE STORAGE TANK
TANK 5
RADIUS 9.25
HEIGHT 23
TANK 4
RADIUS 9.25'
HEIGHT 23
PENTACHLOROPHENOL STORAGE TANK
BASIN
INV. L: 103.90
110
111
112
109

Facility Layout

Figure 3.0

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C. ENFORCEMENT HISTORY

In 1981, following passage of CERCLA, one of the former owners of a portion of the Site notified EPA under CERCLA Section 103, that hazardous waste may have been handled at the Site.

On July 2, 1982, the FDER inspected the Site and took samples for analysis. A number of organic compounds were detected by analysis of those samples. In December 1982 EPA completed a Hazard Ranking System scoring of the Site and, subsequently, placed the Site on the National Priority List.

Contractors for both the EPA and the James Graham Brown Foundation carried out investigations at the Site in 1983. AMAX Environmental Services, Inc., also independently reviewed the Site investigations during that time.

On September 14, 1983, the EPA issued an administrative order requiring that an RI/FS Work Plan be prepared. In response to the order, AMAX Environmental Services, Inc. and the James Graham Brown Foundation (AMAX/Brown) agreed to undertake an RI/FS study. On March 22, 1984, an RI/FS Work Plan was submitted to EPA and the FDER by AMAX/Brown. This Work Plan was incorporated by reference into the EPA Consent Order (for an RI/FS) dated April 20, 1984 and the parallel FDER Consent Order filed on April 19, 1984.

At a meeting with the EPA and the FDER on February 22, 1985, AMAX/Brown was asked to prepare a revised RI/FS Work Plan along with a new schedule for the completion of the Work Plan. The Work Plan was resubmitted and the tasks therein implemented. The RI/FS was completed in August 1987.

A summary of the site investigations is presented in Table 1.0.

In October 1987 a meeting between EPA and AMAX/Brown occurred. The discussion included the introduction by EPA of the possibility for a removal of lagoon sludges by AMAX/Brown during the winter dry season. In November 1987 AMAX/Brown stated their intention to do a removal of the lagoon sludges. EPA approved of AMAX/Brown's proposed activities and began negotiating a Consent Order while the removal developed. The Consent Order was completed in January 1988 and the removal activities were completed in February 1988. AMAX/Brown have been very cooperative in furthering the cleanup of the site.

Table 1.0

Summary of Site Investigations

Company/Agency	Date	Activities
Florida DER	July 2, 1982	Private well sampling/ site inspection
NUS Corporation	February 9, 1983	Private well, surface water and soil sampling
EPA Emergency Response Team	June 1983	Surface soil/waste sampling [1]
LETCO	September 1983	Soil borings/ground water/surface water sampling
LETCO	November 1984	Preliminary Site Investigation Report [2]
FTC&H/EEM	March 1986	Draft RI Report [3]
ReTeC	August 1986	Lagoon test pits/ TCLP analyses
FTC&H/EEM	March, 1987	Final RI report [4]
ERT	July 1987	Risk Assessment [5]

[1] P.E. LaMoreaux and Associates Inc. "A Hydrogeologic Evaluation of the Impact of Past Waste Disposal Operations at an Abandoned Wood Preserving Plant in Live Oak, Florida and Recommendations for Remedial Action". November 15, 1983.

[2] Law Engineering and Testing Company "Final Report of Investigation for the Brown Wood Preserving Site, Live Oak, Florida". November 20, 1984.

[3] FTC&H and Environmental Engineering Management "Report on the Remedial Investigation, Brown Wood Preserving Site, Live Oak, Florida". (First draft) March 6, 1986.

[4] FTC&H and Environmental Engineering Management "Report on the Remedial Investigation, Brown Wood Preserving Site, Live Oak, Florida". (Final)

[5] ERT "Risk Assessment to Accompany the Feasibility Study of the Live Oak Superfund Site, Live Oak, Florida". July 1987.

D. COMMUNITY RELATIONS HISTORY

Residents near the Site are generally aware that the Site was a wood processing facility sometime in the past and that it is a hazardous waste site.

The administrative record was installed in the Site repository in Live Oak, Florida, on September 29, 1987. A notice regarding the administrative record and a future public meeting was placed in the local newspaper on October 1, 1987. The public comment period began on November 25, 1987 and ended on December 16, 1987. The public meeting on the RI/FS results and the presentation of the remedy of choice took place on December 9,1987 in Live Oak, Florida. The meeting was attended by very few local citizens.

The EPA Remedial Project Manager received no comments from the public on the proposed remedy or on any other facet of the project. However, reports from the FDER's local liaison and from a local newspaper reporter indicate that the community is pleased that EPA, the FDER, and AMAX/Brown moved so rapidly to cleanup the site. There are no environmental activist groups involved at the site.

E. ALTERNATIVES EVALUATION

1. ALTERNATIVE DEVELOPMENT

The National Contingency Plan (40 CFR 300.68) specifies that remedial alternatives should be classified as either management of migration (off-site migration) or source control.

Management of migration remedial action as specified in 40 CFR 300.68(e)(3) is necessary where hazardous substances have migrated from the original source of contamination and pose a significant threat to public health, welfare or the environment. Management of migration remedial actions have been eliminated by the feasibility study because the Remedial Investigation concluded that the contaminants deposited at the Site have remained in place and do not pose an immediate danger to human health, welfare or the environment.

Source controls as defined in 40 CFR 300.68(e)(2) address situations in which "a substantial concentration of hazardous substances remain at or near the area where they were originally located and inadequate barriers exist to retard migration of substances into the environment." Source control remedial actions may include alternatives to contain the hazardous substances in place or eliminate potential contamination by transporting the hazardous substances to a new location. Based upon the above definition, the purpose of source control remedial actions is to prevent or minimize the migration of hazardous substances from the Brown Wood Preserving Site. In order to facilitate the development of alternatives, technologies are arranged by general category and applicability in Table 2.0. From the above list of technically feasible remedial action technologies, seven (7) specific alternatives were developed for the Brown Wood Site. These alternatives are described in Table 7.0.

In addition to the above requirements for the development of alternatives based on technical feasibility, the U.S. EPA Guidance on Feasibility Studies under CERCLA (June 1985) states: "At least one alternative for each of the following must, at a minimum, be evaluated within the requirements of the feasibility study guidance and presented to the decisionmaker:

(a) Alternatives for treatment or disposal at an offsite facility approved by EPA (including RCRA, TSCA, CWA, CAA, MPRSA, and SDWA approved facilities), as appropriate;

(b) Alternatives which attain applicable and relevant Federal public health or environmental standards;

(c) As appropriate, alternatives which exceed applicable and relevant public health or environmental standards;

(d) Alternatives which do not attain applicable or relevant public health or environmental standards but will reduce the likelihood of present or future threat from the hazardous substances. This must include an alternative which closely approaches the level of protection provided by the applicable or relevant standards and meets CERCLA's objective of adequately protecting public health, welfare and environment.

(e) A no-action alternative."

2. ALTERNATIVE SCREENING PROCESS

The purpose of the initial screening process is to identify, develop, and incorporate complementary mitigating technologies into site-specific alternatives. The National Oil and Hazardous Substances Contingency Plan (NCP) Section 300.68(g)(h) outlines the process for developing and screening remedial alternatives. The NCP states "a limited number of alternatives should be developed for either source control or offsite remedial action (or both) depending upon the type of response that has been identified." Furthermore, "the alternatives developed under CFR 300.68(g), Development of Alternatives, will be subjected to an initial screening to narrow the list of potential remedial actions for further detailed analysis." Three broad criteria should be used in the initial screening of alternatives: 1) cost; 2) effects of the alternatives; and 3) acceptable engineering practice. In accordance with CFR 300.68(g) and (h) and U.S. EPA Guidance on Feasibility Studies Under CERCLA, the initial screening process of remedial action technologies was divided into six (6) steps:

- Identification of Remedial Action Technologies based upon General Response Actions;
- Development of Technological Feasibility Criteria and Screening (acceptable engineering practice);
- Development of Remedial Action Alternatives;
- Development of Environmental and Public Health Criteria and Screening (acceptable engineering practice);
- Other Criteria Screening; and
- Cost Estimating and Screening.

The technologies/alternatives remaining after the initial screening process were subjected to a detailed evaluation.

3. ALTERNATIVE TECHNOLOGIES

Several alternative technologies were studied for possible utilization as a remedy. The seven (7) technologies considered were: in-situ containment, source removal, water treatment, biological treatment, thermal treatment, solvent extraction, and disposal. These seven (7) technologies were further specified into twenty-four (24) separate specific technological alternatives.

a. In-situ containment technologies were eliminated from consideration because of the risk of a partial sinkhole collapse in the lagoon and because the method would result in no reduction in the toxicity or volume of the contaminants.

b. Several source removal technologies were evaluated. The technologies evaluated in this category apply to sludge and heavily contaminated soils found in the lagoon, the discharge ditch and the plant area. Included in this category are inflow control, water removal, and shallow excavation. There were no significant impediments to implementing these technologies at the Site. Source removal will be a necessary component of most of the remedial action alternatives evaluated.

c. The water treatment technologies evaluated are applicable to contaminated water removed from the lagoon as part of the source removal action. Technologies considered included: spray irrigation, evaporation, treatment and discharge to a surface stream, and pretreatment and discharge to the municipal sewer. The last option is considered the most feasible option from a technical, economic and administrative perspective.

d. Biological treatment technologies considered included: in-situ treatment, biological batch reactors and land treatment. In-situ treatment is technically feasible for low level contaminated soil. However, the majority of the wastes requiring treatment are highly contaminated sludges. Therefore, in-situ treatment of highly contaminated sludges was excluded. Biological batch reactors are potentially feasible for pretreatment of the highly contaminated sludges. The residues from this process would still require final treatment and disposal. Land treatment of low-level wastes is considered feasible and was retained as an alternative technology.

e. Thermal treatment technologies included: examination of the Huber System, the SHIRCO incinerator, the mobile rotary kiln, the mobile circulating fluidized bed, the commercial incinerator, and the industrial kiln. These technologies are applicable to the sludges and the contaminated soils. Technologies evaluated included both mobile incinerators (for on-site waste incineration) and stationary, off-site commercial incineration facilities. The SHIRCO system is considered the most technically feasible option. This system will be evaluated further in the review of the remedial action alternatives. Several industrial kilns and commercial incinerators are technically feasible. Of these options, the Marine Shale industrial kiln in Louisiana is considered the most economically viable and was retained for further evaluation.

Table 2.0

SUMMARY OF TECHNOLOGY SCREENING

TECHNOLOGY	APPLICABLE
Containment Technologies	
o In-situ solidification	No
o Vertical Seepage Cutoffs	No
o Horizontal Barriers	No
Source Removal	
o Water Inflow Control	Yes
o Water Removal	Yes
o Excavation	Yes
Water Treatment Technologies	
o Pretreatment and Municipal Discharge	Yes
o Treatment and Surface Water Discharge	No
o Evaporation	No
o Spray Irrigation	Yes
Biological Treatment Technologies	
o Land Treatment	Yes
o Batch Reactors	Yes[1]
o In-situ Treatment	No[2]
Thermal Treatment Technologies	
o Huber System	No
o SHIRCO	Yes
o Mobile Rotary Kiln	No
o Mobile Circulating Fluidized Bed	No
o Commercial Incinerator	No
o Industrial Kiln	Yes
Solvent Extraction	
o B.E.S.T. Process	No
o Critical Fluid Extraction System	No
o PCB Soil Decontamination Process	No
Disposal	
o On-site Vault	No
o Off-site Commercial Facilities	Yes

[1] Only for sludges

[2] Applicable to low level contaminated soils

f. Three solvent extraction processes were considered: the B.E.S.T. process, the critical fluid extraction system, and the PCR soil decontamination process. These technologies are applicable to low-level sludges as well as highly contaminated sludges. There is a lack of operating history with these technologies. The most advanced system for which there is a commercially available unit is the B.E.S.T. process. However, this alternative was eliminated due to (1) the extensive research and development work which would be required, (2) the extensive utility requirements for the unit, (3) the need to find a use for the oils recovered from the process, and (4) the uncertain regulatory status of the system.

g. Two (2) disposal alternatives were examined: (1) on-site vaulting and (2) disposal at off-site commercial facilities. Permanent on-site vaulting was eliminated due to RCRA land disposal regulations, the Site's location in karst terrain, and Florida's ban on the siting of hazardous waste landfills within the State. Disposal at off-site commercial facilities was retained for further evaluation.

4. SUMMARY OF ALTERNATIVES EVALUATIONS

As described in Table 2.0 and above, seven (7) general cleanup technologies further divided into twenty-four (24) remedial alternatives were initially screened with the intent to reduce the number of alternatives to be evaluated in detail. This initial screening process involved the use of several criteria: 1) technical feasibility; 2) public health effects; 3) environmental effects; 4) attainment of the legally applicable or relevant and appropriate requirements of other Federal and State public health or environmental laws, or provides the grounds for invoking one of the six waivers provided for in SARA; 5) site-specific application; and 6) cost.

Section 121 (b)(1) and (b)(2) of CERCLA/SARA says:

(1) Remedial actions in which treatment which permanently and significantly reduces the volume, toxicity or mobility of the hazardous substances, pollutants, and contaminants is a principal element, are to be preferred over remedial actions not involving such treatment. The offsite transport and disposal of hazardous substances or contaminated materials without such treatment should be the least favored alternative remedial action where practicable treatment technologies are available. The President shall conduct an assessment or permanent solutions and alternative treatment technologies or resource recovery technologies that, in whole or in part, will result in a permanent and significant decrease in the toxicity, mobility, or volume of the hazardous substance, pollutant, or contaminant. In making such assessment, the President shall specifically address the long-term effectiveness of various alternatives. In assessing alternative remedial actions, the President shall, at a minimum, take into account:

(A) the long-term uncertainties associated with land disposal;
(B) the goals, objectives, and requirements of the Solid Waste Disposal Act;
(C) the persistence, toxicity, mobility, and propensity to bioaccumulate of such hazardous substances and their constituents;
(D) short- and long-term potential for adverse health effects from human exposure;
(E) long-term maintenance costs;
(F) the potential for future remedial action costs if the alternative remedial action in question would fail; and
(G) the potential threat to human health and the environment associated with excavation, transportation, and redisposal, or containment.

The President shall select a remedial action that is protective of human health and the environment, that is cost-effective, and that utilizes permanent solutions and alternative treatment technologies or resource recovery technologies to the maximum extent practicable. If the President selects a remedial action not appropriate for a preference under this subsection, the President shall publish an explanation as to why a remedial action involving such reductions was not selected.

(2) The president may select an alternative remedial action meeting the objectives of this subsection whether or not such action has been achieved in practice at any other facility or site that has similar characteristics. In making such a selection, the President may take into account the degree of support for such remedial action by parties interested in such site.

Of the twenty-four (24) specific alternatives, fourteen (14) were eliminated in the initial screening. The remaining ten (10) were merged into seven (7) and the seven (7) specific alternatives were subjected to detailed screening procedures (Table 2.0 describes the results of the process.). A more detailed evaluation of the seven (7) specific cleanup alternatives follows.

a. NO-ACTION

The no-action alternative implies leaving the site in its present condition without disturbing the contaminated materials. This alternative has the advantage that both the short-term and the long-term risks of exposure are not impacted from their present levels. The risk of spread of contaminated materials is not increased. A part of the no-action alternative may be continued monitoring of the groundwater allowing identification of any changes in site conditions or the migration of contaminated materials off-site. Should changes be discovered which increase the risks associated with this site, this alternative could be reassessed and, if necessary, alternative actions could be taken.

b. ON-SITE INCINERATION

This alternative includes excavation of all contaminated materials at the site followed by on-site incineration. The steps required for this alternative include:

1. site preparation including mobilization and establishment of on-site support facilities;
2. inflow control including construction of engineered berms and drainage ditches;
3. water removal from the lagoon to site storage tanks and other water disposal system;
4. discharge to the POTW of water from site storage tanks and other water disposal system after treatment, if necessary;
5. excavation of lagoon sludges and contaminated soils and short haul to incinerator staging area;
6. incineration by use of the SHIRCO incinerator on-site;
7. site restoration including regrading to establish engineered drainage patterns and appropriate revegetation.

Institutional requirements for this alternative include obtaining local approvals for discharge to the POTW and completing a pilot test burn to demonstrate the applicability of the technology in meeting RCRA and State air quality performance standards.

The impacts of this alternative include elevated risks for short-term exposure to construction workers and process employees. The impacts of these risks can be reduced, as discussed above, by implementation of a site-specific health and safety plan. There is no long-term risk associated with any contaminated materials as they would be detoxified on-site and delistable.

Prior to implementation of this alternative, a pilot test burn would be required to prove the viability of the treatment technology and to define system operating parameters. Based upon the results of the pilot test, burn performance objectives would be established. Once these steps are completed, the alternative could be implemented relatively quickly.

The time to accomplish the on-site incineration alternative is an estimated minimum of seven (7) months.

No long-term operation, maintenance, and monitoring requirements other than continued groundwater monitoring are associated with this alternative.

c. LAND TREATMENT

This alternative assumes that all the contaminated materials at the site are excavated and land-treated in the former railroad tie storage area. Prior to implementation of this alternative, an on-site pilot scale demonstration would be necessary using the actual waste and site soils. Following this demonstration, a detailed engineering design for the facility would be prepared. The demonstration test would take approximately six months to one year to complete. An additional six months would be necessary to prepare a treatment demonstration work plan on the front end and a detailed design on the back end.

Implementation of this alternative would involve the following steps:

1. site preparation including mobilization, establishment of on-site support facilities, and construction of the land treatment area (which would include grading, berm and ditch construction and construction of a site perimeter fence and road);
2. inflow control;
3. lagoon water removal, treatment (if necessary), and disposal;
4. excavation and waste spreading according to the design plan and schedule;
5. soil incorporation and cultivation using a rototiller device;
6. soil treatment for at least one year;
7. closure, including providing a vegetative cover and post-closure care (including groundwater, soil, and soil pore water monitoring) for up to thirty (30) years unless the zone of incorporation could be delisted.

The initial excavation and waste application period could occur within three months of initiating site activities. The treatment period would extend for one to two years.

The primary institutional requirement for this alternative involves completing an extensive demonstration test which would simulate full-scale operating conditions.

d. OFF-SITE INCINERATION

This alternative includes excavation of all contaminated materials at the site and transportation to an industrial process kiln, where the materials would be processed into construction aggregate. The steps required with this alternative include:

1. site preparation including mobilization and establishment of on-site support facilities;
2. inflow control;
3. lagoon water removal, treatment (if necessary), and disposal;
4. excavation of lagoon sludges and contaminated soils, stabilization of at least a portion of the lagoon sludge;
5. transportation of excavated materials to an industrial process kiln where they would be processed by incineration into asphalt aggregate; and
6. site restoration.

All transportation loads must be manifested and carried by licensed hazardous waste haulers. Permits for the industrial process kiln would be verified prior to initiating the process. However, the responsibility for obtaining and maintaining the necessary permits for the incinerator is that of the owner/operator.

The impacts of this alternative include elevated risks for short-term exposure to site workers. In addition, there is some risk to the general population associated with transportation of the materials. There is no long-term risk associated with any contaminated materials as they would be detoxified and delisted.

It is estimated that this alternative would take at least seven (7) months to implement completely. No long-term operation and maintenance requirements except groundwater monitoring are associated with this alternative.

e. TREATMENT AND OFF-SITE DISPOSAL

Implementation of this technology as a remedial action alternative would require the following steps:

1. site preparation;
2. inflow control;
3. lagoon water removal, treatment (if necessary), and disposal;
4. excavation, treatment (if necessary) by a mechanical or pozzolanic stabilization process of lagoon sludges and contaminated soils;
5. disposal of treated and untreated materials at an EPA-approved disposal site; and
6. site restoration.

Institutional considerations for this alternative include obtaining approval from local agencies for discharge to the local POTW. The permanent status of the disposal site must be verified and commercial disposers must accept these contaminated materials. The licenses of the hazardous waste transporter must be verified and waste manifests prepared prior to shipment.

For this alternative ther are elevated risks for short-term exposure mainly to site workers. There is also a short-term risk to the general population associated with hauling of the contaminated materials. At least four (4) months would be required to complete the excavation, removal, and restoration alternative.

This alternative would remove all contaminated materials from the site, so no long-term operation and maintenance requirements except groundwater monitoring would be incurred.

f. TREATMENT AND DISPOSAL OF SLUDGES AND LAND TREATMENT OF SOILS

This alternative assumes that the sludges are treated on-site using either mechanical treatment or stabilization and disposed off-site; and the contaminated soils are land-treated on-site. This alternative is a combination of the land treatment and off-site disposal alternatives It offers more flexibility than either alternaitve as the most highly contaminated sludges are immediately removed from the site. The soils of low-level contamination are temporarily stored while a treatment demonstration is completed.

The basic steps of this alternative are:

1. site preparation;
2. inflow control;
3. lagoon water removal, treatment (if necessary), and disposal;
4. sludge excavation, treatment (where necessary), and removal to an EPA-approved disposal facility;
5. soil excavation and storage in several lined and temporarily capped cells;
6. land treatment area construction on-site on approximately fourteen (14) acres;
7. soil spreading over land treatment area;
8. soil incorporation with nutrients, etc.;
9. treatment; and
10. closure.

Institutional considerations for this alternative include obtaining approval from local agencies for discharge to the POTW and completing a land treatment demonstration.

Assuming a one-year period for the demonstration and design phase, the treatment could begin within one year of initiating sludge removal activities. As the application would occur in a single batch process, the entire waste application could be completed within one month and the treatment period would last approximately two (2) years.

Post-closure activities could last for thirty (30) years.

g. BIOLOGICAL TREATMENT OF SLUDGES AND LAND TREATMENT

This alternative assumes that the sludges are pretreated biologically using sequenced batch reactors followed by land treatment of the biosludge and contaminated soils. This alternative would significantly reduce the organic content of the sludges thereby decreasing land area requirements for the land treatment alternative.

The pretreatment process would use three (3) polyethylene lined reactors, each approximately 250,000 gallons in capacity and equipped with three (3) 25-horsepower agitator/aerator units. The system would be operated in the plug flow mode with an average solids residence time of seven (7) days/reactor. The resulting solids from this process would be hauled to the land treatment area for final treatment. Vendors of this technology suggest that up to 90% reductions in total creosote constituents would be produced by this process.

This alternative would take a minimum of nine (9) months to complete. It would require completion of a land treatment demonstration and laboratory treatability studies to determine the final design parameters for the sludge biological pretreatment process. The basic steps and schedule identified in the land treatment alternative apply to thios alternative as well except that the land area requirements for the pretreated sludges decrease to about six (6) acres.

Table 3.0

Estimated Volumes of Contaminated Materials

(Prior to removal which occurred Dec.'87 - Feb.'88 under EPA approval)

Location	Material	Area (ft^2)	Depth (ft)	Volume (yd^3)
Plant Area	Soil	40600(1)	0.5(3)	750-1100
Ditch	Sludge	3000(1)	3.5(4)	400-600
Lagoon	Sludge	106938(2)	0-4.0(4)	3000
Lagoon Soil	Soil	106938(2)	0-3(5)	1000-6500

Notes:

(1) Area calculated from "Residue Area", Fishbeck-Thompson Carr & Huber, Inc. "Amax/Brown Wood Preserving Site Hydrologic Study", Sheet 10, Project No. F 84816A.

(2) Law Engineering Testing Co., "Final Report of Investigation for the Brown Wood Preserving Site, Job No. GE4271, dated November 1984.

(3) Assumed average depth based on analytical results sample numbers SL-055, 058, 061, 062, 067, 300-323, 400, 401.

(4) Assumed depth based on Test Hole 12 by Ardaman and Associates, 1985, and Test Pit 3 by ERT, 1985.

(5) See text for assumptions

Table 4.0

Contaminant Concentrations Below Samples
With Total Creosote Concentrations >1000 ppm

Sample Description		Total Creosote Concentration (ppm)		Carcinogenic PAH's (ppm)		% Carcinogenic PAH's	
		above	below	above	below	above	below
February 1983 Soil Samples (Table A.4)	Drainage Ditch	210300		17500		8%	
	Surface Impoundment	398300		21300		5%	
June 20-24, 1983 Ditch Soil Samples (Table A.5)	A-01, 2 - 2.5 ft	2548		781		31%	
	B-01	25566		6927		27%	
	C-01, 2 ft	70776-186910		1875 -27400		3-15%	
	D-01, 0.5 - 1.5 ft	28672-179769		719 -60879		3-34%	
	D-02, 2 - 2.5 ft	5139		1006		20%	
	E-01, 0.5 - 1.5 ft	2563		895		35%	
	F-01, 0.5 - 1.5 ft	1052		19		2%	
	G-01, 1.4 - 2.2 ft	1357		29		2%	
	H-01, 0.5 - 1.5 ft	4414		1426		32%	
	I-07, 2 - 2.5 ft	10750		1540		14%	
	J-05, 3.6 ft	5764-199030		111 -24000		2-12%	
June 20-24, 1983 Lagoon Soil Samples (Table A.5)	K-02, 0 - 1.6 ft	27764		5171		19%	
	K-03, 0 - 0.5 ft	134153		15960		12%	
	L-02A, 0 - 1.0 ft	3648		1132		31%	
	1 - 1.8 ft		2667		164		6%
	L-06A, 0 - 2.6 ft	1014		372		37%	
	2.8 - 3.1 ft		50.1		24		48%
	L-07, 0 - 1.5 ft						
September 1983 Soil Borings (Table A.7)	A-6, 0 - 1.0 ft	1005		269		27%	
	2 - 3 ft		71		24		34%
January 1985 Soil Test Hole Samples From Lagoon Vicinity (Table A.9)	Test Hole 5, 0 - 1 ft	1615		594		37%	
	3 - 5 ft		0.34		0		0%
	Test Hole 7, 0 - 1 ft	3360		690		21%	
	3 - 8 ft		1.05		0		0%
	Test Hole 8, 0 - 1 ft	16300		0		0%	
	3 - 8 ft		116.2		10.8		9%
	Test Hole 9, 0 - 1 ft	37390		3800		10%	
	3 - 5 ft		4.31		0		0%
	Test Hole 10, 0 - 1 ft	5345		800		15%	
	3 - 5 ft		13.11		0.15		1%
From Plant Site Vicinity (Table A.10)	Test Hole 12, 0 - 1 ft	146389		11600		8%	
	5 - 7 ft		166.9		8.63		5%
	Test Hole 13, 0 - 1 ft	16770		900		5%	
	5 - 7 ft		157.2		6.65		4%
	11 - 13 ft		1966		88.5		5%
	Test Hole 14, 0 - 1 ft	2713		918		34%	
	9 - 11 ft		1009		118.2		12%
August 1986 Soil Test Pits in Lagoon (Table A.15)	Test Pit 3, 0 - 0.5 ft	101110		3980		4%	
	Test Pit 2, 1 - 3 ft		218		15.9		7%

Table 5.0
CHRONOLOGY OF ENVIRONMENTAL SAMPLING
LIVE OAK, FLORIDA

	Date	Sampled	n	For
FDER	Summer/82	surface and ground water (private wells)	4	"purgeable organics"
EPA	2/8/83	air		
EPA	2/8/83	ground water (private wells)	7	metals
EPA	2/9/83	surface water	3	creo. constituents, pcp
EPA	2/83	sediment (ditch and lagoon)	2	creo. constituents, pcp
EPA/PELA	6/83	borings		creo. constituents, pcp
PELA	6/24/83	surface water	3	creo. constituents, pcp
LETCO	9/83	borings	7	creo. constituents, pcp
EEM	9/84	surface soil (plant area)	7	creo. constituents, pcp
EEM	1/85	borings (ditch and plant)	10	creo. constituents, pcp
EEM	8/85	surface soil (plant)	26	creo. constituents, pcp
EEM	8/85	surface soil (wood storage)	16	creo. constituents, pcp
EEM	9/85	ground water (monitoring wells)	5	creo. constituents, pcp
EEM	10/86	ground water (monitoring well)	7	creo. constituents, pcp
EEM	1/87	ground water (monitoring well and private well)	3	creo. constituents

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*creo. constituents = phenol, dibenzofuran and PAH associated with creosote.

*pcp = pentachlorophenol

Table 6.0

INDICATOR CHEMICALS

Compound	Weight of Evidence[a]	Solubility[b]	Log Kow[c]
Benzo (a) Anthracene	B2	14	5.61
Benzo (b) Fluoranthene	B2	0.8	6.06
Benzo (a) Pyrene	B2	3.8	6.06
Chrysene	B2	2	5.61
Dibenzo (a,h) Anthracene	B2	0.5	6.77
Indeno (1,2,3,cd) pyrene	C	0.2	
Fluoranthene	not ranked	260	4.90
Pentachlorophenol	D	14000	5.01

(a) EPA (1986). There are only a limited number of chemical compounds that have been demonstrated unequivocally to be human carcinogens. However, experimental and epidemiologic data are available that are suggestive of the carcinogenic activity of certain compounds. The EPA "weight-of-evidence" system for ranking from A to D (in decreasing order) the level of certainty that a compound is a human carcinogen is explained in the text. There are no A or B-1 level carcinogens present at the Live Oak Site. Certain PAH compounds present at the site have been rated at B-2 or C-level potential carcinogens. To obtain an appropriately conservative risk assessment the assumption is made that compounds are human carcinogens, even if there is limited certainty that this effect is real. As such, potential carcinogens down to level C weight of evidence have been selected. (EPA, 1986).

(b) Solubility (in ppb), at 25°C. Data for PAH from Craun and Middleton, 1984, data for pentachlorophenol from EPA, 1980c.

(c) Log octanol/water coefficient. Data for PAH from EPA, 1980b. Data for pentachlorphenol EPA, 1980c.

F. SELECTED REMEDY DESCRIPTION AND RATIONALE FOR SELECTION

1. EXTENT OF REMOVAL ACTIVITY

An EPA-approved removal occurred in December 1987 through March 1988. This removal was undertaken by AMAX/Brown in accordance with an Administrative Consent Order completed in January 1988. The removal included:

a. Mobilization, including establishment of a site office and on-site safety zones;
b. Removal of lagoon water to allow excavation of sludges;
c. Excavation, treatment, and disposal of approximately 15,000 tons of creosote contaminated lagoon sludge and soil at the Chemical Waste Management, Inc. (CWM) secure landfill near Emelle, Alabama;
d. Treatment of approximately 200,000 gallons of lagoon water removed as part of the lagoon excavation;
e. Extensive sampling and analyses of soil and water at appropriate locations and times; and
f. Dismantling, decontamination, and disposal of the old wood preserving plant in an EPA-approved manner.

More detailed information on the removal is contained in Attachment A ("LIVE [illegible] INTERIM REMOVAL" Memorandum, John Ryan, February 11, 1988).

2. SELECTED REMEDY

The selected remedy embodies the remaining work necessary to complete the remediation of the site after considering the work accomplished by the removal activities in December 1987 through March 1988.

The recommended alternative is basically Alternative f. : Treatment and Disposal of Sludges and Land Treatment of Soils. However, there are four modifications to Alternative f.: (1) if the land treatment (biodegradation) does not attain the desired cleanup levels for the appropriate organic contaminants within the time allowed, then an alternative means of dealing with the contaminated soils, such as removal, incineration, solidification, or vitrification, will be determined by EPA at that time; (2) ground water monitoring will continue for five years; (3) contaminated soils exceeding certain higher contaminant levels may be stabilized and removed to an EPA-approved hazardous waste facility (Emelle, Alabama or Pinewood, South Carolina); and (4) contaminated soils exceeding certain lower contaminant levels will be biodegraded in an onsite excavated area that has been lined.

The remedy is consistent with 40 CFR Part 300.68 (J) in that the above-modified Alternative f. is technically feasible, alleviates all existing and potential health effects, presents no new public health hazards and substantially reduces the threat to the surface and ground water and the general environment.

Preference is given to this option because of technical feasibility, cost, site-specific permanence, and the existence of Land Disposal Restriction variances which allow its implementation within certain advantageous time frames. Modification of the processes involved in this option may be required in order to satisfy design requirements and site conditions.

Creosote, the wood preservative, consists of approximately two hundred (200) different compounds. The below-mentioned compounds are six (6) of those two hundred. These compounds were selected as indicators because the EPA weight-of-evidence system indicates that they are "possible" or "probable" human carcinogens.

Benzo [a] anthracene
Benzo [a] pyrene
Benzo [b] fluoranthene
Chrysene
Dibenzo [a,h] anthracene
Indeno [1,2,3,c,d] pyrene

Note: Fluoranthene and pentachlorophenol were also considered.

The selection of indicator parameters is based upon numerous previous priority pollutant analyses conducted during the Remedial Investigation phase. Although other types of contaminants were present onsite, these compounds are among the most common and potentially the most carcinogenic found at the Brown Wood Site.

Cleanup standards were based upon the results of a Risk Assessment which focussed on attaining at least a 1 x 10-6 risk with regard to ingestion of contaminated soil by a child. Cleanup standards were described by means of the total concentration of the six indicator parameters.

A detailed cost development and analysis of selected remedial alternatives was done prior to the removal activity (prior to December 1987) to assure that the most cost-effective remedial action was chosen for the Brown Wood Site. Cost estimates followed the procedures specified in 40 CFR 300.68 (8)(2)(B), Guidance on Feasibility Studies under CERCLA, and the Remedial Action Costing Procedures Manual.

Twenty-four (24) specific remedial action alternatives underwent the evaluation process. Fourteen (14) were eliminated on the basis of site-specific application, technical feasibility, public health and welfare, and environmental evaluations. The ten (10) remaining were merged into seven (7). A detailed cost analysis was performed for each of the remaining seven (7) alternatives. These alternatives are listed in Table 7.0.

Results of the Detailed Screening of Alternatives

(Prior to the removal which was conducted Dec.'87 thru Feb.'88)

	Alternatives						
Effectiveness	No Action	On-Site Inciner.	Land Treat.	Off-Site Inciner.	Off-Site Disposal	Sludge Treatment & Land Treatment	Biological Pretreat & Land Treatment
Protective of Public Health	Below	Average	Average	Average	Average	Average	Average
Attains ARARs	Below	Above	Average	Above	Average	Average	Average
Reduces TMV	Below	Above	Average	Above	Below	Average	Average
Minimal Envir. Impacts	Below	Average	Average	Average	Average	Average	Average
Reliable	Above	Average	Average	Below	Average	Average	Average
Implementability							
Feasibility & Availability	Above	Below	Average	Above	Above	Above	Average
Constructable	Above	Below	Above	Above	Above	Above	Above
Timeliness	Below	Average	Average	Above	Above	Above	Average
O&M Requirement	Below	Average	Average	Above	Above	Average	Average
Administrative Feasibility	Average	Below	Below	Average	Above	Average	Below
Costs ($000)							
Capital Cost	0	5,440	1,900	11,300	4,140	2,740	2,400
Annual O&M Cost	20	0	0	0	0	0	0
Total Present Value Cost	190	5,440	1,900	11,300	4,140	2,740	2,400

The final remedy consists of three (3) major tasks.

a. Site preparation which will include the following activities:

1) Clearing, grubbing, and grading the proposed biological treatment area, where necessary;
2) Installing a drainage swale in the appropriate locations(s);
3) Installing a perimeter fence around the land treatment area; and installing signs on the perimeter fence warning against exposure to hazardous material.

b. Construction of the Treatment System for biodegradation which will include the following tasks:

1) Site grading;
2) Installing a liner throughout the treatment area;
3) Installing a drainage system on top of the liner and underneath the soils to be treated;
4) Installing a spray irrigation system within the land treatment areas;
5) Constructing a stockpile/holding area for the soils to be treated;
6) Installing a water aeration system within the land treatment area;
7) Hooking up utilities; and
8) Spreading contaminated soil and consolidating the stockpile.

c. Operation and Maintenance

1) Tilling, irrigation, and fertilization of the land treatment area;
2) Maintenance of the water/leachate treatment system;
3) Monitoring of the land and water treatment systems;
4) Semi-annual monitoring of the ground water quality for five (5) years after completion of construction activities; review of site's condition after five (5) years (See Section 121(c), CERCLA/SARA.); and
5) Maintenance of security fences and warning signs.

3. CLEANUP STANDARDS

The cleanup standards for the selected remedy are based upon the Risk Assessment and addenda and were finalized by EPA after discussions with the FDER and AMAX/Brown. These standards are describeable by referring to one factor: the total concentration of carcinogenic creosote constituents (indicators).

a. Standards for soils treated in the old lagoon and new land treatment area:

Within two (2) years from its initial seeding the land treatment process must reduce the concentration of total carcinogenic indicator chemicals to 100 parts per million (ppm) throughout the volume of the material treated. This level for total carcinogenic indicator chemicals corresponds to an approximate 1 x 10-6 soil ingestion risk level. Upon successful completion of the bioremediation in the land treatment area, the land treatment area shall be revegetated.

b. Standards for the plant area, the wood storage area and other site areas:

Upon completion of the remedial activities in the plant area, the wood storage area, and any other site areas, the soils must contain no more than a 100 ppm total for the carcinogenic indicator chemicals.

4. RATIONALE FOR SELECTION OF FINAL REMEDY

The removal described in F.1. above, according to engineering calculations, caused the majority of the contamination (i.e., the contaminated sludges and soils in and around the lagoon) to be properly disposed of at the CWM facility in Emelle, Alabama, or in Pinewood, South Carolina. The selected remedy described in F.2. above comprises the major activities necessary to complete remediation of the site in a technically feasible and cost-effective manner consistent with CERCLA/SARA the NCP, and applicable or relevant and appropriate requirements (ARAR's).

With regard to EPA's decision to enter into an Administrative Consent Order with AMAX/Brown for the removal described in F.1. above, Sections 104(a)(2) indicates that any removal action undertaken by either the President (EPA) or by potentially responsible parties with the President's (EPA's) approval should "contribute to the efficient performance of any long term remedial action" with respect to the release or threatened release concerned. Section 122 (e)(6) indicates that once a remedial investigation and feasibility study has been initiated "no potentially responsible party may undertake any remedial action at the facility unless such remedial action has been authorized by the President" (e.g., EPA authorizes activity by means of a Consent Order).

The removal activities not only eliminated the major source of contamination and set the stage for the final site remediation activities, but contributed to the acceleration of the site along the Superfund enforcement process track. The final remedy consists of those remaining activities which will bring the site into compliance with those cleanup standards described in the Risk/Health Assessment and addenda (See Attachment B) and approved by both EPA and the State of Florida.

The selected remedy is a combination of two general source control remedial actions as defined in 40 CFR 300.68(e)(2). These two measures are: 1) removal to the CWM facility in Emelle, Alabama, or in Pinewood, South Carolina, of the most severely contaminated soils/sludges; and 2) onsite biodegradation of the less severely contaminated soils to achieve the cleanup levels indicated in the Risk/Health Assessment. In case number one, the governing regulatory factor has been the RCRA Land Disposal Restriction (LDR) phase-in. The LDR phase-in schedule has allowed the removal of creosote contaminated sludges and soils thus eliminating potential onsite contamination by transporting the hazardous substances to a secure hazardous waste landfill at a different location. In case number two, an alternative and innovative technology, biodegradation, is to be used on less severely contaminated soils to "polish" those soils to acceptable levels of cleanliness. EPA's Office of Solid Waste and Emergency Response (OSWER), the Office of Technology Assessment (OTA), and the Office of Research Development (ORD) have, in response to CERCLA/SARA, recognized that our ability to characterize or assess the extent of contamination, the chemical and physical character of the contaminants, or the stresses imposed by the contaminants on complex ecosystems is limited, and new, innovative technologies are needed. The Brown Wood Preserving Site now provides EPA with an excellent opportunity to utilize and re-assess biodegradation in a controlled situation in EPA's Region IV. The selected remedy dictates that the bioremediation will occur in a lined area which is designed with interior drainage and spray irrigation systems. The land treatment area will also have a perimeter security fence and an exterior drainage system.

III. RESPONSIVENESS SUMMARY

A. BACKGROUND ON COMMUNITY INVOLVEMENT

Historically, community concern regarding the Brown Wood Preserving Site has been low, according to EPA, the FDER, local officials, and the local news media. There have been no recorded complaints from the local residents. On the contrary, the community seems genuinely glad that EPA and the FDER have acted rapidly to clean up the site.

B. SUMMARY OF PUBLIC COMMENTS AND AGENCY RESPONSES

There were no comments from the public during or after the public comment period. An article indicating the time and place for the public meeting was placed in the Suwannee Democrat newspaper more than two weeks prior to the actual meeting date. Few people attended the public meeting and there were only three or four general questions which had no bearing on the selected remedy. Additionally, the administrative record required under Section 113 of CERCLA/SARA was placed in the site repository given below on September 29, 1987:

Suwannee River Regional Library
ATTN: Ms. Faye Roberts
Reference Librarian
207 Pine St.
Live Oak, Florida 32060
(904) 362-2317

Self-addressed and stamped envelopes were left with the administrative record. The first draft of the Record-of-Decision and an instruction sheet for commenters was also deposited with the other records. However, the EPA Regional Project Manager received no mail or telephone calls from the community regarding either the proposed remedy or the actual onsite activities.

The Agency has responded to the low level of interest by periodically speaking with newspaper reporters who are with the Suwannee Democrat. Newspaper atricles followed these telephone and in-person discussions with the reporters.

C. EXPLANATIONS OF DIFFERENCES BETWEEN PROPOSED PLAN AND THE SELECTED REMEDY

The proposed plan in the Feasibility Study is generally the same as the selected remedy in the Record-of-Decision. There are basically five differences in the two: (1) the removal of lagoon sludges has already occurred; (2) the grubbing of the majority of the land treatment area has already occurred; (3) waters pumped from the lagoon prior to the excavation of the sludges have already been treated and properly disposed of; (4) the land treatment area will be lined; and (5) the cleanup standards with regard to the original Risk Assessment were changed.

D. COMMUNITY RELATIONS CONDUCTED AT THE SITE PRIOR TO AND DURING THE PUBLIC COMMENT PERIOD

From 1984 through 1987 EPA, FDER, and AMAX/Brown representatives maintained contact with Live Oak City officials while undertaking the Remedial Investigation / Feasibility Study. Numerous newspaper articles appeared in the local Suwannee Democrat describing the onsite activities. Several City Council meetings were attended and the community was made aware of EPA, FDER, and PRP activities.

The administrative record was installed in the site repository in September, 1987, and through the Suwannee Democrat the community was made aware of the progress made towards the cleanup. In November through the middle of December, 1987, the public comment period occurred. In December the public meeting occurred. There were no public comments forthcoming from the community. Only comments of a specific, technical nature were received from the PRPs' contractor.

ATTACHMENT A

Memorandum on the Extent of Removal Activity which occurred December 1987 through February 1988

RETEC
REMEDIATION
TECHNOLOGIES INC

MEMO TO: Distribution
FROM: J. Ryan
DATE: February 11, 1988

RE: LIVE OAK INTERIM REMOVAL

INTRODUCTION

This memorandum summarizes activities completed at the Live Oak site as part of interim removal activities during the period December, 1987 through the present. These activities have included:

- mobilization,
- removal of lagoon water to allow excavation of sludges,
- excavation, treatment and disposal of sludges and contaminated soil at the CWM secure landfill, Emelle, AL, and
- treatment of the lagoon water removed as part of excavation.

Extensive sampling and analyses of soil and water were completed during these activities.

MOBILIZATION

Mobilization occurred the first week in December. An office trailer and decontamination trailer were brought to the site as well as the necessary excavation and pumping equipment. A health and safety plan was reviewed with the site workers and the site separated into "clean" and "contaminated" zones for truck staging and decontamination purposes.

Diversion ditches were excavated around the perimeter of the lagoon to divert run on to the north. The location of these ditches are shown on Figure 1. A sedimentation trap was installed in the diversion ditches consisting of a series of hay bales placed across the ditch.

REMOVAL OF LAGOON WATER

Standing water was pumped from the lagoon using a vacuum truck. This water was stored in four former product storage tanks on site with an estimated capacity of 250,000 gallons. Once all standing water was removed, a series of trenches were excavated in the low part of the lagoon to continue dewatering the lagoon while excavation was ongoing. The vacuum truck continued to remove the free liquids while the excavation was underway.

EXCAVATION, TREATMENT AND DISPOSAL

The lagoon sludges were treated with kiln dust in place by mixing the dust with the sludge using power shovels. When a portion of the lagoon was stabilized, it was excavated and staged on the sides of the lagoon for subsequent loading onto the transport vehicles.

Sampling of the upper four feet of soil was conducted in the lagoon by excavating test pits with a trackhoe and collecting samples from the pit walls. Only soil was sampled (no sludge) to identify the depth of contamination that exceeded 1000 ppm of total creosote substances (TCS). Figure 1 shows the location of the sampling points and the attached tables summarize the analytical results. These results are prior to sludge excavation. In general, no contaminated soil was found on the western end of the lagoon whereas the eastern end had significant contamination.

Table 1 presents a daily log of materials transported to Emelle. A total of 15,000 tons of contaminated material was hauled from the site between December and the end of January. This total includes over 6000 tons of sludges which exceeded 100,000 ppm TCS. The remainder was highly contaminated soils greater than 5,000 ppm TCS.

Low level contaminated sand and some of the underlying clays were also excavated. These soils are currently stockpiled around the eastern end of the lagoon. It is estimated that these soils represent approximately 10,000 tons of materials. Table 3 presents data from various samples collected from the lagoon and the stockpiled soils which represent the average composition of these stockpiled soils. The concentration of TCS ranges from less than 1000 ppm to 5000 ppm with an average around 3000 ppm. Carcinogenic PAH range from 100 to 200 ppm. It is this stockpiled material which will be treated biologically.

Approximately one foot of native clays were backfilled in the lagoon and compacted in place to provide a contoured surface. The lowest part of the lagoon is now approximately 10 feet lower than its original surface. Standing water covers the bottom to a depth of approximately two feet. This water is run-off and has an oily sheen. Figure 1 shows the current elevations of the bottom of the lagoon.

TREATMENT OF WATER

The water which was removed from the lagoon to facilitate excavation was stored in four tanks in the process area. In addition, approximately 70,000 gallons of water were pumped out of the retort pit. Approximately 200,000 gallons of water were pumped into these tanks. Table 4 presents analyses of the water prior to treatment.

The initial attempt to treat the water indicated that the water would clog the filters due to a strong emulsion. Subsequently a flocculation step was added to break the emulsion. The current treatment system consists of flocculation, followed by sand filtration followed by a micron filter followed by carbon adsorption. The treated effluent is then discharged to a series of four temporary storage tanks, (20,000 gallons capacity per tank) for sampling purposes. The stored effluent is then spray irrigated over a three acre area on-site located to the west of the plant.

The initial 40,000 gallons treated were sampled for volatiles, semi-volatiles and copper chromium and arsenic. Results of this sampling are noted below and the complete laboratory results are presented as Table 5.

	Tank #1	Tank #2
2 Butanol	39 ppb	ND
Phenanthrene	5 ppb	ND
Fluoranthene	5 ppb	ND
Pyrene	3 ppb	ND
Arsenic	<5 ppb	<5 ppb
Copper	3 ppb	3 ppb
Chromium	13 ppb	6 ppb

These results were transmitted verbally to Dr. Walker of the FDER on Monday, February 1, 1988 and Ms. Danner, of U.S. EPA, on Tuesday February 2. Both individuals gave permission to spray irrigate the water. Ms. Danner also requested that we continue to sample each tank for PAH and report the results prior to spraying. Tanks 1, 3, and 4 were subsequently analyzed for PAH compounds and these results were verbally transmitted to Dr. Walker on Monday, February 8. All the PAH compounds were below detection levels of two parts per billion in Tanks 3 and 4. Tank 1 had detectable quantities of certain PAH compounds.

Fluoranthene was detected at 40 parts per billion and pyrene was detected at 29 parts per billion. The remaining compounds were less than 20 parts per billion. Dr. Walker gave verbal approval to spray irrigate the water on Tuesday. To date, 100,000 gallons of water have been spray irrigated. Treatment of the water will continue until the contents of the tanks are emptied. It is then proposed to proceed with the dismantling of the plant as described in the attached memorandum concerning a conceptual plan for the final remedy.

ATTACHMENT A

TABLE 1

TRANSPORTATION LOG SUMMARY
LIVE OAK, FLORIDA

DATE	NET TONS
12-15	304.11
12-16	602.13
12-17	473.45
12-18	698.73
12-19	517.13
12-20	322.61
1-4	1097.25
1-5	1233.27
1-6	1298.27
1-7	494.38
1-8	437.00
1-9	324.97
1-10	1318.33
1-11	1623.51
1-12	619.50
1-13	527.30
1-14	291.33
1-15	136.10
1-18	167.18
1-19	643.39
1-20	430.67
1-21	639.79
1-22	595.72
1-23	238.92
1-26	22.51
TOTAL	15057.55

TABLE 2

PAH CONCENTRATIONS IN LAGOON SOIL

PAGE 1

COMPOUND	SAMPLE, CONCENTRATIONS IN ug/g (ppm)					
	2-3A	2-3B	2-3C	3-5A	3-5B	3-5C
NAPHTHALENE	160	0.96	0	20	0	0
2-METHYL NAPHTHALENE	120	0	0	0	0	0
ACENAPHTHYLENE	0	0	0	0	0	0
ACENAPHTHENE	100	1	0	0	0	0
FLUORENE	89	0	0	0	0	0
PHENANTHRENE	180	0	0	0	0	0
ANTHRACENE	0	0	0	0	0	0
FLUORANTHENE	150	0	0	0	0	0
PYRENE	110	0	0	0	0	0
BENZO(A) ANTHRACENE	14	0	0	0	0	0
CHRYSENE	0	0	0	0	0	0
BENZO(B) FLUORANTHENE	3	0	0	0	0	0
BENZO (K)FLUORANTHENE	0	0	0	0	0	0
BENZO(A)PYRENE	0	0	0	0	0	0
INDENO(1,2,3-CD) PYRENE	0	0	0	0	0	0
DIBENZO(A,H)ANTHRACENE	0	0	0	0	0	0
BENZO(GHI)PERYLEN	0	0	0	0	0	0
TOTAL PAH	926	1.96	0	20	0	0
CARCINOGENIC PAH	17	0	0	0	0	0

TABLE 2

PAH CONCENTRATIONS IN LAGOON SOIL

PAGE 2

COMPOUND SAMPLE, CONCENTRATIONS IN ug/g (ppm)

COMPOUND	4-6A	4-6B	5-3A	5-3B	6-1A	6-1B
NAPHTHALENE	130	130	0	0.6	0	0
2-METHYL NAPHTHALENE	44	120	0	0	0	0
ACENAPHTHYLENE	0	0	0	0	0	0
ACENAPHTHENE	18	140	0	0	0	0
FLUORENE	25	110	0	0	0	0
PHENANTHRENE	36	180	6.7	6.8	0	0
ANTHRACENE	0	0	0	0	0	0
FLUORANTHENE	9	120	3	3.5	0	0
PYRENE	9	100	0	1.5	0	0
BENZO(A) ANTHRACENE	0	13	0	0	0	0
CHRYSENE	0	0	0	0	0	0
BENZO(B) FLUORANTHENE	0	0	0	0	0	0
BENZO (K)FLUORANTHENE	0	0	0	0	0	0
BENZO(A)PYRENE	0	0	0	0	0	0
INDENO(1,2,3-CD) PYRENE	0	0	0	0	0	0
DIBENZO(A,H)ANTHRACENE	0	0	0	0	0	0
BENZO(GHI)PERYLEN	0	0	0	0	0	0
TOTAL PAH	271	913	9.7	12.4	0	0
CARCINOGENIC PAH	0	13	0	0	0	0

TABLE 2

PAH CONCENTRATIONS IN LAGOON SOIL

PAGE 3

COMPOUND	SAMPLE, CONCENTRATIONS IN ug/g (ppm)					
	6-2A	6-2B	6-3A	6-3B	7-1A	7-1B
NAPHTHALENE	0	0	0	0	0	0
2-METHYL NAPHTHALENE	0	0	0	0	0	0
ACENAPHTHYLENE	0	0	0	0	0	0
ACENAPHTHENE	0	0	0	0	0	0
FLUORENE	0	0	0	0	0	0
PHENANTHRENE	0	0	0	0	0	0
ANTHRACENE	0	0	0	0	0	0
FLUORANTHENE	0	0	0	0	0	0
PYRENE	0	0	0	0	0	0
BENZO(A) ANTHRACENE	0	0	0	0	0	0
CHRYSENE	0	0	0	0	0	0
BENZO(B) FLUORANTHENE	0	0	0	0	0	0
BENZO (K)FLUORANTHENE	0	0	0	0	0	0
BENZO(A)PYRENE	0	0	0	0	0	0
INDENO(1,2,3-CD) PYRENE	0	0	0	0	0	0
DIBENZO(A,H)ANTHRACENE	0	0	0	0	0	0
BENZO(GHI)PERYLEN	0	0	0	0	0	0
TOTAL PAH	0	0	0	0	0	0
CARCINOGENIC PAH	0	0	0	0	0	0

TABLE 2

PAH CONCENTRATIONS IN LAGOON SOIL

PAGE 4

COMPOUND	SAMPLE, CONCENTRATIONS IN ug/g (ppm)			
	7-2A	7-2B	7-3A	7-3B
NAPHTHALENE	0	0	0	0
2-METHYL NAPHTHALENE	0	0	0	0
ACENAPHTHYLENE	0	0	0	0
ACENAPHTHENE	0	0	0	0
FLUORENE	0	0	0	0
PHENANTHRENE	0	0	0	0
ANTHRACENE	0	0	0	0
FLUORANTHENE	0	0	0	0
PYRENE	0	0	0	0
BENZO(A) ANTHRACENE	0	0	0	0
CHRYSENE	0	0	0	0
BENZO(B) FLUORANTHENE	0	0	0	0
BENZO (K)FLUORANTHENE	0	0	0	0
BENZO(A)PYRENE	0	0	0	0
INDENO(1,2,3-CD) PYRENE	0	0	0	0
DIBENZO(A,H)ANTHRACENE	0	0	0	0
BENZO(GHI)PERYLEN	0	0	0	0
TOTAL PAH	0	0	0	0
CARCINOGENIC PAH	0	0	0	0

TABLE 2

PAH CONCENTRATIONS IN LAGOON SOIL

PAGE 5

COMPOUND	SAMPLE, CONCENTRATIONS IN ug/g (ppm)					
	2-1A	2-1B	2-2A	2-2B	3-1A	3-1B
NAPHTHALENE	46	2700	1000	90	860	500
2-METHYL NAPHTHALENE	23	1800	470	64	350	470
ACENAPHTHYLENE	0	0	0	0	0	0
ACENAPHTHENE	72	1100	580	62	330	250
FLUORENE	17	1600	560	110	340	380
PHENANTHRENE	13	1500	1200	64	530	340
ANTHRACENE	0	280	31	0	22	0
FLUORANTHENE	4.6	940	940	86	440	320
PYRENE	1.9	680	660	61	320	140
BENZO(A) ANTHRACENE	0	150	150	0	55	0
CHRYSENE	0	150	187	24	44	90
BENZO(B) FLUORANTHENE	0	46	64	0	26	11
BENZO (K)FLUORANTHENE	0	0	0	0	0	0
BENZO(A)PYRENE	0	75	41	0	12	0
INDENO(1,2,3-CD) PYRENE	0	0	0	0	0	0
DIBENZO(A,H)ANTHRACENE	0	0	0	0	0	0
BENZO(GHI)PERYLEN	0	0	0	0	0	0
TOTAL PAH	177.5	11021	5883	561	3329	2501
CARCINOGENIC PAH	0	421	442	24	137	101

TABLE 2

PAH CONCENTRATIONS IN LAGOON SOIL

PAGE 6

COMPOUND	SAMPLE, CONCENTRATIONS IN ug/g (ppm)	
	5-1A	5-1B
NAPHTHALENE	15	0
2-METHYL NAPHTHALENE	0	0
ACENAPHTHYLENE	0	0
ACENAPHTHENE	0	0
FLUORENE	5.3	0
PHENANTHRENE	4.6	3.6
ANTHRACENE	0	0
FLUORANTHENE	0	0
PYRENE	0	0
BENZO(A) ANTHRACENE	0	0
CHRYSENE	0	0
BENZO(B) FLUORANTHENE	0	0
BENZO (K)FLUORANTHENE	0	0
BENZO(A)PYRENE	0	0
INDENO(1,2,3-CD) PYRENE	0	0
DIBENZO(A,H)ANTHRACENE	0	0
BENZO(GHI)PERYLEN	0	0
TOTAL PAH	24.9	3.6
CARCINOGENIC PAH	0	0

TABLE 3

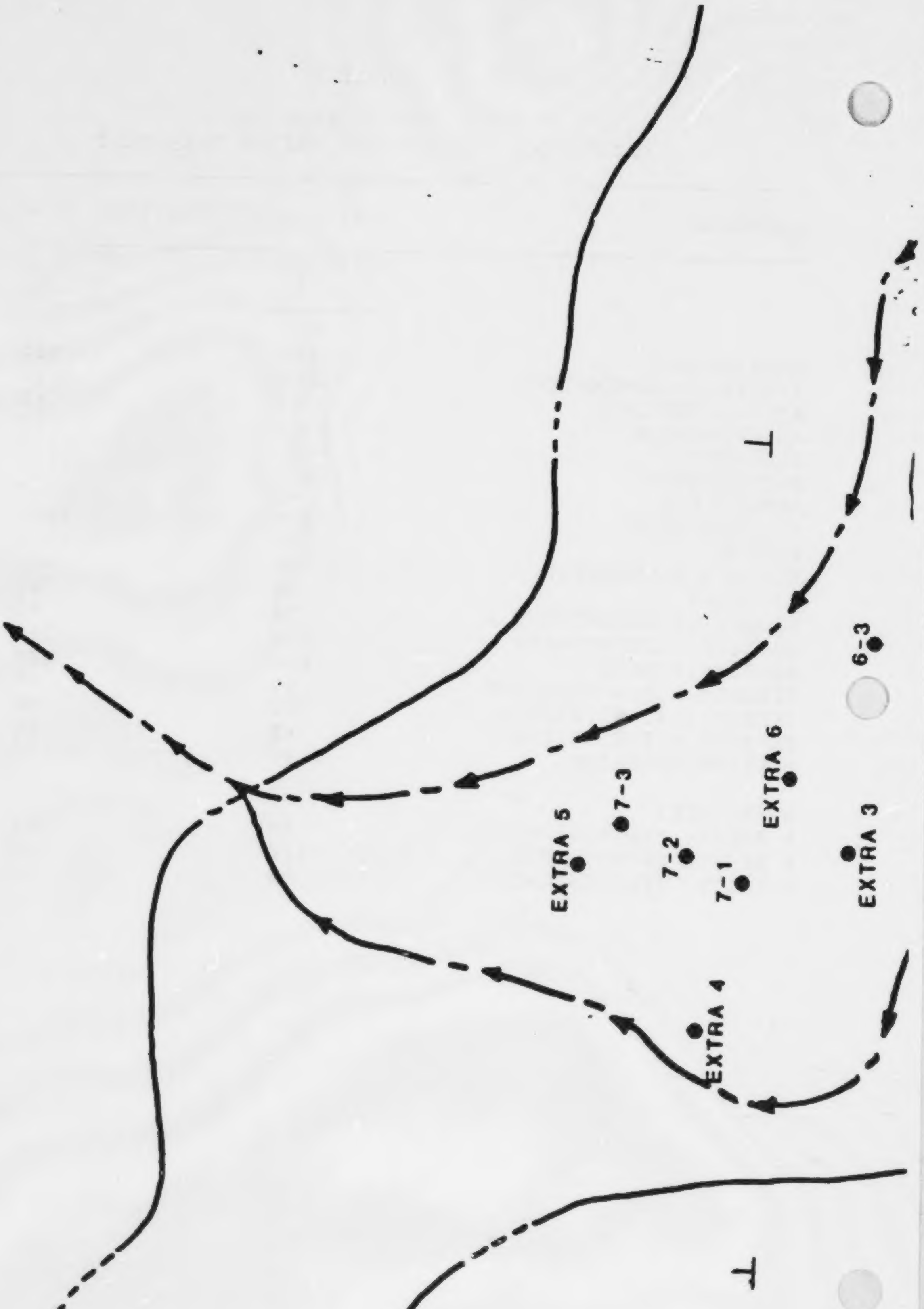
CONCENTRATIONS OF PAH COMPOUNDS IN SOILS TO BE LAND TREATED AT LIVE OAK SITE

COMPOUND	SAMPLE CONCENTRATIONS IN mg/Kg (ppm)							
	SPOILS PILE 3-1	SPOILS PILE LO-2	SURFACE SOIL LO-1	SPOILS PILE 1-1	3-1A	3-1B	AVERAGE	MAX
NAPHTHALENE	1100	1800	1200	<3	860	500	910	1800
2-METHYL NAPHTHALENE	410	650	480	<3	350	470	393	650
ACENAPHTHYLENE	<3	<3	<3	<3	<2	<2	0	0
ACENAPHTHENE	230	420	280	41	330	250	259	420
FLUORENE	250	270	270	43	340	380	259	380
PHENANTHRENE	1500	170	340	81	530	340	494	1500
ANTHRACENE	<3	<3	44	56	22	<2	20	56
FLUORANTHENE	330	190	470	420	440	320	362	470
PYRENE	290	74	200	320	320	140	224	320
BENZO (A) ANTHRACENE	42	23	39	48	55	<2	35	55
CHRYSENE	63	45	40	96	44	90	63	96
BENZO (K) FLUORANTHE	32	41	25	32	<2	<2	22	41
BENZO (B) FLUORANTHE	12	<3	7	29	26	11	14	29
BENZO (A) PYRENE	18	28	23	35	12	<2	19	35
DIBENZO (A,H) ANTHRA	8	4	3	<3	<2	<2	3	8
INDENO (1,2,-CD) PYR	<3	<3	<3	<3	<2	<2	0	0
BENZO (G,H,I) PERYLE	3	5	2	<3	<2	<2	2	5
TOTAL PAH	4288	3720	3423	1201	3329	2501	3077	4288

TABLE 4

ANALYTICAL RESULTS:
LAGOON (OR POND) WATER BEFORE TREATMENT

COMPOUND	SAMPLE CONCENTRATIONS IN ug/l (ppb)	
	INFLUENT #1	INFLUENT #2
NAPHTHALENE	20500	560
2-METHYL NAPHTHALENE	2700	180
ACENAPHTHYLENE	<5	<5
ACENAPHTHENE	5100	920
FLUORENE	4600	1200
PHENANTHRENE	10600	9300
ANTHRACENE	<5	<5
FLUORANTHENE	3800	4800
PYRENE	2100	4000
BENZO(A)ANTHRACENE	32~~0~~	400
CHRYSENE	370	720
BENZO(K)FLUORANTHENE	56	40
BENZO(B)FLUORANTHENE	210	360
BENZO(A)PYRENE	400	480
DIBENZO(A,H)ANTHRACENE	<5	<5
INDENO(1,2,-CD)PYRENE	<5	<5
BENZO(G,H,I)PERYLENE	<5	<5
PENTACHLOROPHENOL	2100	<1000
SURROGATES		
% RECOV. FLUOROBIPHENYL	34	52
% RECOV. TRIBROMOPHENOL	120	110
% RECOV. TERPHENYL-D14	55	98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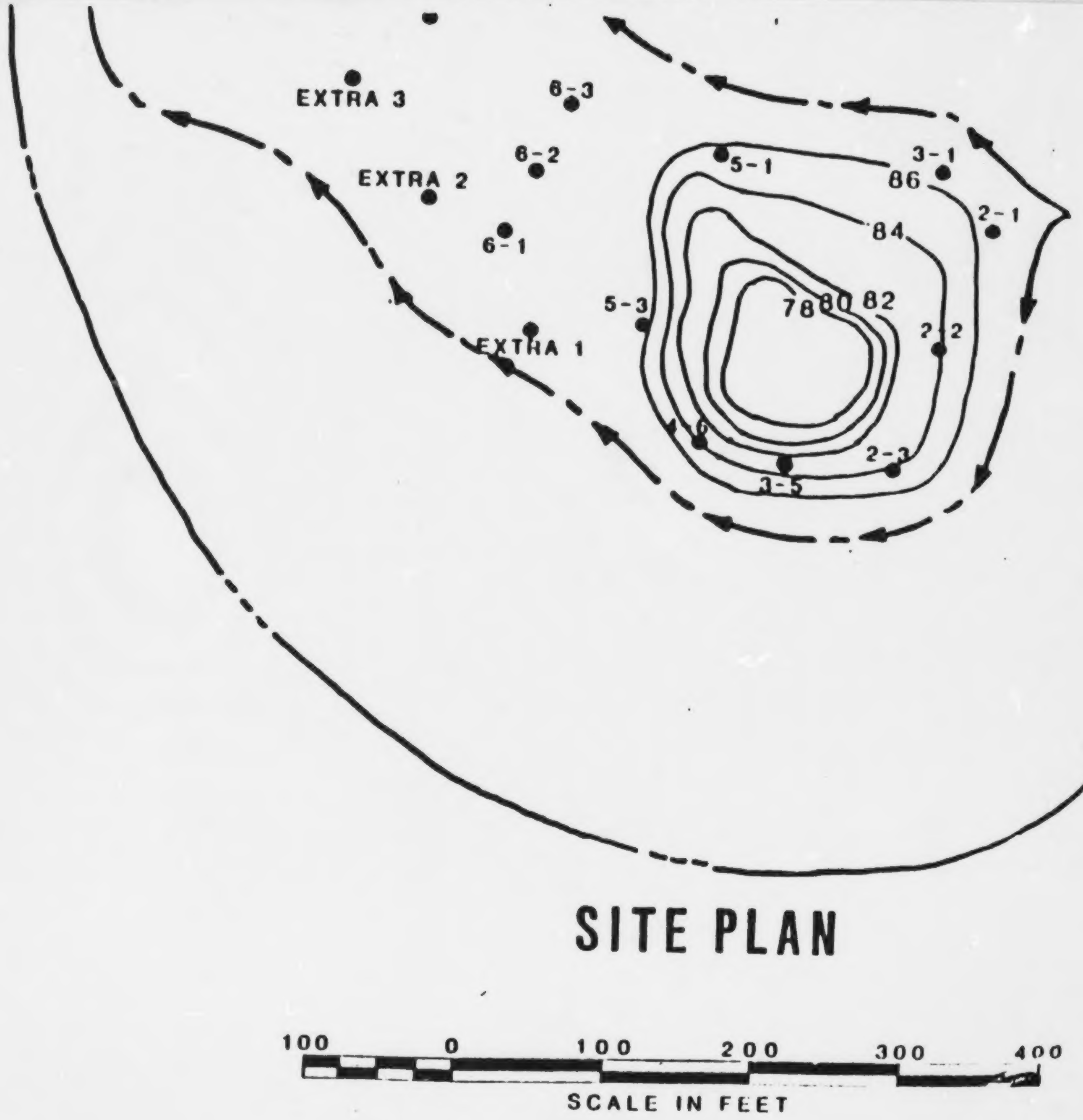
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86
2-1
84
78
80
82
2-2
2-3
3-5
100
0
100
200
300
400
SCALE IN FEET

SITE PLAN

FIGURE 1

ATTACHMENT A

1

Laucks
Testing Laboratories, Inc.
940 South Harney St. Seattle Washington 98108 (206)767-5060

Certificate

Chemistry Microbiology and Technical Services

CLIENT: Remediation Technology, Inc.
19219 West Valley Hwy., Suite M103
Kent, WA 98032
ATTN: John Ryan

LABORATORY NO. 8028

DATE: Feb. 2, 1988

Proj. No. C86-006-910

REPORT ON: WATER

SAMPLE IDENTIFICATION: Submitted 01/29/88 and identified as shown:

1) Water #1 01/28/88 16:15
2) Water #2 01/28/88 16:30

TESTS PERFORMED AND RESULTS:

	parts per million (mg/L)	
	1	2
Arsenic	<0.005	<0.005
Copper	0.003	0.003
Chromium	0.013	0.006

Samples were analyzed in accordance with Test Methods for Evaluating Solid Waste, (SW-846), U.S.E.P.A., 1982, Method 8270 (semi-volatile extractables).

Extractables (by GC/MS)

	parts per billion (ug/L)		
	1	2	Lab Blank
Phenol	<2.	<2.	<2.
*Aniline	<10.	<10.	<10.
Bis(2-Chloroethyl)Ether	<2.	<2.	<2.
2-Chlorophenol	<2.	<2.	<2.
1,3-Dichlorobenzene	<2.	<2.	<2.

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Laucks
Testing Laboratories, Inc.
940 South Harney St Seattle Washington 98108 (206)767 5060
Chemistry Microbiology and Technical Services

Certificate

PAGE NO. 2

Remediation Technology, Inc.

LABORATORY NO. 8028

parts per billion (ug/L)

	1	2	Lab Blank
1,4-Dichlorobenzene	<2.	<2.	<2.
*Benzyl Alcohol	<2.	<2.	<2.
1,2-Dichlorobenzene	<2.	<2.	<2.
*2-Methylphenol	<2.	<2.	<2.
Bis(2-Chloroisopropyl)Ether	<2.	<2.	<2.
*4-Methylphenol	<2.	<2.	<2.
N-Nitroso-Di-n-Propylamine	<2.	<2.	<2.
Hexachloroethane	<4.	<4.	<4.
Nitrobenzene	<2.	<2.	<2.
Isophorone	<2.	<2.	<2.
2-Nitrophenol	<2.	<2.	<2.
2,4-Dimethylphenol	<2.	<2.	<2.
*Benzoic Acid	<50.	<50.	<50.
Bis(2-Chloroethoxy)Methane	<2.	<2.	<2.
2,4-Dichlorophenol	<4.	<4.	<4.
1,2,4-Trichlorobenzene	<2.	<2.	<2.
Naphthalene	<4.	<4.	<4.
*4-Chloroaniline	<2.	<2.	<2.
Hexachlorobutadiene	<2.	<2.	<2.
4-Chloro-3-Methylphenol	<4.	<4.	<4.
*2-Methylnaphthalene	<2.	<2.	<2.
Hexachlorocyclopentadiene	<4.	<4.	<4.
2,4,6-Trichlorophenol	<4.	<4.	<4.
*2,4,5-Trichlorophenol	<4.	<4.	<4.
2-Chloronaphthalene	<2.	<2.	<2.
*2-Nitroaniline	<4.	<4.	<4.
Dimethyl Phthalate	<2.	<2.	<2.
Acenaphthylene	<2.	<2.	<2.
*3-Nitroaniline	<10.	<10.	<10.
Acenaphthene	<2.	<2.	<2.
2,4-Dinitrophenol	<20.	<20.	<20.

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PAGE NO. 3

Remediation Technology, Inc.

LABORATORY NO. 8028

parts per billion (ug/L)

	1	2	Lab Blank
4-Nitrophenol	<20.	<20.	<20.
*Dibenzofuran	<2.	<2.	<2.
2,4-Dinitrotoluene	<4.	<4.	<4.
2,6-Dinitrotoluene	<2.	<2.	<2.
Diethyl Phthalate	<2.	<2.	<2.
4-Chlorophenyl-Phenylether	<2.	<2.	<2.
Fluorene	<2.	<2.	<2.
*4-Nitroaniline	<4.	<4.	<4.
4,6-Dinitro-2-Methylphenol	<20.	<20.	<20.
N-Nitrosodiphenylamine	<2.	<2.	<2.
1,2-Diphenylhydrazine	<4.	<4.	<2.
4-Bromophenyl-Phenylether	<4.	<4.	<4.
Hexachlorobenzene	<2.	<2.	<2.
Pentachlorophenol	<20.	<20.	<20.
Phenanthrene	5.	<2.	<2.
Anthracene	<2.	<2.	<2.
Di-n-Butyl Phthalate	<2.	<2.	<2.
Fluoranthene	5.	<2.	<2.
Pyrene	3.	<2.	<2.
Benzidine	<50.	<50.	<50.
Butylbenzylphthalate	<2.	<2.	<2.
3,3'-Dichlorobenzidine	<20.	<20.	<20.
Benzo(a)Anthracene	<2.	<2.	<2.
Bis(2-Ethylhexyl)phthalate	<2.	<2.	<2.
Chrysene	<2.	<2.	<2.
Di-n-Octyl Phthalate	<2.	<2.	<2.
Benzo(b)Fluoranthene	<4.	<4.	<4.
Benzo(k)Fluoranthene	<4.	<4.	<4.
Benzo(a)Pyrene	<4.	<4.	<4.
Indeno(1,2,3-cd)Pyrene	<4.	<4.	<4.
Dibenzo(a,h)Anthracene	<4.	<4.	<4.
Benzo(g,h,i)Perylene	<4.	<4.	<4.

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PAGE NO. 4

LABORATORY NO. 8028

Remediation Technology, Inc.

Samples were analyzed in accordance with Test Methods for Evaluating Solid Waste (SW-846), U.S.E.P.A., 1982, Method 8240 (volatile organics).

Volatile Organics (by GC/MS)

	parts per billion (ug/L)		
	1	2	Lab Blank
Chloromethane	<1.	<1.	<1.
Bromomethane	<1.	<1.	<1.
Vinyl Chloride	<1.	<1.	<1.
Chloroethane	<3.	<3.	<3.
Methylene Chloride	<1.	<1.	<1.
*Acetone	<5.	<5.	<5.
*Carbon Disulfide	<1.	<1.	<1.
1,1-Dichloroethene	<1.	<1.	<1.
1,1-Dichloroethane	<1.	<1.	<1.
trans-1,2-Dichloroethylene	<1.	<1.	<1.
cis-1,2-Dichloroethene	<1.	<1.	<1.
Total-1,2-Dichloroethene	<1.	<1.	<1.
Chloroform	<1.	<1.	<1.
*2-Butanone	39.	<3.	<3.
1,2-Dichloroethane	<1.	<1.	<1.
1,1,1-Trichloroethane	<1.	<1.	<1.
Carbon Tetrachloride	<1.	<1.	<1.
*Vinyl Acetate	<1.	<1.	<1.
Bromodichloromethane	<1.	<1.	<1.
1,2-Dichloropropane	<1.	<1.	<1.
Trichloroethene	<1.	<1.	<1.
Benzene	<1.	<1.	<1.
Chlorodibromomethane	<3.	<3.	<3.
1,1,2-Trichloroetnane	<1.	<1.	<1.
Bromoform	<1.	<1.	<1.

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PAGE NO. 5

Remediation Technology, Inc.

LABORATORY NO. 8028

parts per billion (ug/L)

	1	2	Lab Blank
*4-Methyl-2-pentanone	<3.	<3.	<3.
*2-Hexanone	<3.	<3.	<3.
1,1,2,2-Tetrachloroethane	<3.	<3.	<3.
Tetrachloroethene	<1.	<1.	<1.
Toluene	<1.	<1.	<1.
Chlorobenzene	<3.	<3.	<3.
trans-1,3-Dichloropropene	<3.	<3.	<3.
Ethylbenzene	<1.	<1.	<1.
cis-1,3-Dichloropropene	<3.	<3.	<3.
*Styrene	<1.	<1.	<1.
*Total Xylenes	<1.	<1.	<1.

Key

* = additional compounds from the EPA's Hazardous Substances List
< = "less than"

Respectfully submitted,

Laucks Testing Laboratories, Inc.

J. M. Owens

JMO:emt

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APPENDIX

Surrogate Recovery Quality Control Report

Attached are surrogate (chemically similar) compounds utilized in the analysis of organic compounds. The surrogates are added to every sample prior to extraction and analysis to monitor for matrix effects, purging efficiency, and sample processing errors. The control limits represent the 95% confidence interval established in our laboratory through repetitive analysis of these sample types.

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CLIENT [illegible] CONTACT [illegible] PROJ.#

PHOENIX ANALYTICAL LABORATORIES, INC.

CHAIN-OF-CUSTODY RECORD

CLIENT PROJECT NO.	PROJECT NAME Live Oak, FL Superfund Site	NO. OF CONTAINERS
SAMPLERS: PRINT NAME [illegible]	SIGNATURE	

CONTAINER TYPE, SIZE

STA. NO.	DATE	TIME	STA. LOC.	SAMPLE TAG #	NO. OF CONTAINERS							ANALYSES REQST'D
Water #1	[illegible]	[illegible]	[illegible]	Water #1	[illegible]	[illegible]	[illegible]	[illegible]				
Water #2	[illegible]	[illegible]	[illegible]	Water #2	6	[illegible]	[illegible]	[illegible]				

no C.C. for John Ryan [illegible] 1/29

Sample Receipt acknowledged pending verification of sample count. You will be notified within one working day of any discrepancies found.
Signed [illegible] Date [illegible] Time [illegible]

RELINQUISHED BY: [illegible] PRINT NAME [illegible] SIGNATURE DAY TIME

RECEIVED BY: Rita Valdez PRINT NAME Lisa Johnson SIGNATURE DAY TIME

RELINQUISHED BY: PRINT NAME SIGNATURE DAY TIME

RECEIVED BY: PRINT NAME SIGNATURE DAY TIME

RELINQUISHED BY: PRINT NAME SIGNATURE DAY TIME

RECEIVED BY: PRINT NAME SIGNATURE DAY TIME

REMARKS INITIALS

ATTACHMENT B

Risk Assessment and addenda

Document No. P-D897

Risk Assessment to Accompany the Feasibility Study of the Live Oak Superfund Site, Live Oak, Florida

Prepared for:

Amax, Inc.
Golden, CO
The James Graham Brown Foundation
Louisville, KY

August 1987

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EXECUTIVE SUMMARY

INTRODUCTION

The following report is a health baseline risk assessment of the Live Oak Superfund Site in Live Oak, Suwannee County Florida. The report follows the guidelines outlined in the Superfund Public Health Assessment Manual (EPA 540/1-86/060) and is intended to accompany the Feasibility Study Report for the site developed by Remediation Technologies Incorporated. A summary of the history of the site and its use as a wood preserving facility is presented in the Feasibility Study Report as well as the Remedial Investigation Report, prepared by Fishbeck, Thompson, Carr and Huber and Environmental Engineering and Management, Limited.

SELECTION OF INDICATOR CHEMICALS

Sampling of media for chemical analysis has been carried out by a large number of agencies and contractors. The chronology of sampling, which occurred between 1982 and 1986 is presented in Table 2-1 of the following report. Most analysis was done with a focus on constituents typically used at wood preserving sites. Creosote constituents and a small amount of pentachlorophenol were found to be present in soil, sediment, and surface water at the site. Certain low molecular weight fractions of creosote were also found in ground water. No metals were found at the site. Based on these findings, creosote constituents and pentachlorophenol were selected as Indicator Chemicals for detailed health risk assessment at the site. The creosote constituents were evaluated as a whole for their potential to produce acute, non-carcinogenic effects on skin. Additionally, six high molecular weight components of creosote were evaluated for potential to produce a carcinogenic response (Benzo[a]anthracene, benzo[a]pyrene, benzo[b]fluoranthene, chrysene, dibenzo[a,h]anthracene, and indeno [1,2,3,c,d,]pyrene). These compounds were selected because the U.S. EPA weight-of-evidence system indicates they are "possible" or "probable" human carcinogens. Another creosote constituent, fluoranthene, was selected for evaluation because it is present in significant amounts at many of the contaminated areas of the site, and because of the existence of an Ambient Water Quality Criterion for this compound. Pentachlorophenol was evaluated for its potential to produce acute, non-carcinogenic effects on skin and also for its potential to produce systemic toxic effects.

EXPOSURE ASSESSMENT

At any site, humans may be exposed to contaminants in air, water, or solid media such as soil and sediments. They may ingest, inhale, or absorb a compound. In certain cases, skin contact without absorption into the system may be considered an exposure. Consideration of the location of the site, its accessibility, and regional hydrogeology indicated that the pertinent routes of potential exposure for the Live Oak site were ingestion of drinking water from the Floridan aquifer if it were to become contaminated, and acute dermal contact with or ingestion of constituents in surface soils by trespassers or visitors to the site.

Concentrations of Indicator Chemicals used in the risk assessment were determined from analytic results or estimated with the aid of mathematical models. The models used for determining potential impact on ground water were a leaching model developed by EPA (the Organic Leaching Model), an analytical one-dimensional unsaturated zone model, and an analytical saturated zone model (the Horizontal Plane Source Model). The models are described in detail in Appendix A of this report. The PAH inputs for the model were obtained from data on sediments and soils in the lagoon area, sampled by the EPA and P.E. LaMoreaux on June 20-24, 1983. Data from the lagoon were used because this is the area with the largest waste load and the most likely place for ground water contamination to occur. The exposure point chosen for the drinking water risk assessment was the nearest downgradient offsite well (Well Number 15 on Sheet 10 of the Remedial Investigation Report). For the direct contact and soil ingestion assessments, on-site surface soil and sediments not normally covered by water were considered.

METHODS USED TO ASSESS RISK

Risk Assessment was carried out using two methods:

- The analytical and predicted concentrations were compared to relevant standards and criteria; and
- Potential intake of Indicator Chemicals using exposure scenarios in which individuals were assumed to drink water from potentially-affected wells or inadvertently ingest surface soils and sediments while visiting the site (the latter scenario is assumed to be relevant to children who soil their hands and put them in their mouth). Potential intakes of non-carcinogenic Indicator Chemicals (PCP and fluoranthene) were compared to "acceptable intakes" developed by the EPA. Potential intakes of the carcinogenic PAH were used to calculate cancer risks using "potency factors" developed by the EPA. For dermal contact, concentrations in the soil were compared to apparent minimal-effects levels determined from the literature.

COMPARISON OF INDICATOR CHEMICAL CONCENTRATIONS TO STANDARDS AND CRITERIA

Comparison of measured or predicted concentrations of Indicator Chemicals in off-site well water to Ambient Water Quality Criteria (the only criteria that appear to be relevant for the Live Oak site) indicate that no impact from pentachlorophenol or fluoranthene is expected. The Ambient Water Quality Criteria for carcinogenic PAH gives a range of values for various risks of cancer. The summed concentration of the carcinogenic PAH predicted to be in the nearest off-site well is less than the concentration associated with a cancer risk of 1 chance in 1,000,000.

ESTIMATION OF CARCINOGENIC RISK

The potency factor for carcinogenic PAH is based on observations of carcinogenic actions of a single compound, benzo[a]pyrene. As this is among the most carcinogenic PAH, using a potency factor based on benzo[a]pyrene for all carcinogenic PAH is conservative. The factor is 0.0115/microgram/kilogram body weight day. This value indicates that an individual taking in one microgram of carcinogenic PAH per kilogram of body weight every day, for life would have a cancer risk of .0115 (i.e. slightly more than one chance in one hundred) in excess of "background" risk.

Using the factor with the predicted intake of carcinogenic PAH that could occur as a result of lifetime ingestion of water in the nearest downgradient well if contamination were to occur indicates that the upper limit cancer risk from this activity is slightly less than 1 chance in 1,000,000. This low risk is often considered "virtually safe."

Using the factor with predicted intake from a less likely exposure scenario, the assumption that children could infrequently visit the site and ingest soils and sediments contaminated with PAH, indicates that an upper-bound cancer risk of eighty eight chances in one hundred thousand.

COMPARISON OF PREDICTED INTAKES TO ACCEPTABLE INTAKES OF NON-CARCINOGENS

Acceptable intakes for pentachlorophenol and fluoranthene are 30 micrograms/kilogram body weight day, and 6.12 micrograms/kilogram body weight day, respectively. These values were developed by the EPA under the assumption that these substances, as non-carcinogens, have a "threshold" for their toxic effects. That is, there is a dose below which virtually no risk of a toxic response exists. The acceptable intakes are assumed to be below the threshold dose for pentachlorophenol and fluoranthene. No estimated intake for pentachlorophenol or fluoranthene using any exposure scenario, was in excess of the acceptable intakes developed by the EPA.

A no-effect level for the dermal effect of creosote and pentachlorophenol was determined by extrapolating from reports in the literature on the photosensitization effects of creosote constituents and the dermal irritation produced by pentachlorophenol. It was determined that concentrations of creosote or pentachlorophenol in excess of 1000 ppm might produce acute dermal effects. Inspection of analytical results indicate that some surface soils and sediments that are not continuously covered with water have concentrations of creosote and pentachlorophenol above 1000 ppm. Thus, some risk of transient dermal effect may exist for the current condition of the site.

DEVELOPMENT OF PERFORMANCE GOALS

For source control remedial alternatives, the Superfund Public Health Evaluation Manual suggests that an analysis to determine "acceptable" levels of residual contamination be performed. No evaluation was deemed necessary for pentachlorophenol or fluoranthene because the baseline assessment indicated no present or future health impacts of the compound. The low level of potential carcinogenic risk from drinking water from off-site also precluded the necessity of performance goals based on this limited health impact. The most likely health impact for the Live Oak site is the potential for non-carcinogenic acute dermal effects from creosote and pentachlorophenol. Thus, performance goals of no more than 1000 ppm creosote or pentachlorophenol in soils or sediments is recommended. If all media containing pentachlorophenol or creosote in excess of these concentrations is removed, it has the secondary effect of decreasing the potential cancer risk produced by inadvertent ingestion of soils and sediments. Using the exposure scenario developed in the baseline risk assessment, the mean concentration of carcinogenic PAH calculated to remain following removal of creosote compounds in excess of 1000 ppm, the cancer risk from ingestion of soils is calculated to be slightly greater than one chance in 1,000,000.

1. INTRODUCTION

The following report is provided to support the asibility Study of the Brown Wood Preserving Site in Live Oak, Florida prepared by Remediation Technologies for Amax, Inc. and The James Graham Brown Foundation, Inc. It is a baseline health risk evaluation, and documentation of the method used for developing performance goals based on human health considerations. The format follows the guidance provided in the Superfund Public Health Evaluation Manual (EPA, 1986).

The baseline evaluation is a health risk assessment of the current condition of the site and, as such, represents a screening of the "no-action alternative." It indicates if a remedial action is needed and how quickly steps must be taken. The methodology developed in the baseline assessment also provides the framework for developing performance goals used for screening remedial alternatives.

The remedial alternatives (other than "No Action") that have been selected for screening for the Live Oak site are source control measures. EPA guidance (EPA, 1986) suggests that such alternatives must be screened for their ability to fulfill requirements of the National Contingency Plan and best engineering judgment. However, as suggested in the Superfund Public Health Evaluation Manual, health-based criteria can be useful in deriving acceptable residual levels of constituents in soils (performance goals). Performance goals will be calculated to provide public health protection at the exposure points identified in the baseline assessment. Specifically, performance goals will be set which would ensure exposure below the Acceptable Intake for Chronic exposure (AIC) for non-carcinogenic toxic constituents and provide for low risk from carcinogenic substances. These values provide an objective, health-based criteria for determining the relative effectiveness of remedial alternatives.

To the extent possible, an analysis of the short term health risks associated with the remedial actions will be provided. Under EPA health assessment guidance (EPA, 1986) this analysis is intended to provide guidance for the health and safety plan accompanying the chosen action.

2. BASELINE ASSESSMENT

The format of this baseline assessment follows the 4-step methodology recommended by the EPA (49 FR 46304, November 23, 1984; 50 FR 1170, January 9, 1985, and EPA, 1986). It should be noted that the terminology used in the Federal Register and the Superfund Public Health Evaluation Manual (SPHEM) is different. Although the differences are only semantic, readers may be familiar with only one terminology. To avoid confusion, the terms and a short description of the methods involved with each step are provided here.

Step 1:	Selection of Indicator Chemicals in the Superfund Public Health Evaluation Manual is comparable to the Hazard Identification in the Federal Register. The assessment reviews the contaminants found in various media. Indicator chemicals are then chosen based on concentration, distribution, toxicity and consistency of detection.
Steps 2 and 3:	Estimation of Exposure Point Concentrations and Estimation of Chemical intakes are comparable to the Exposure Assessment mentioned in the Federal Register. The section reviews the potential exposure pathways, compares relevant standards and criteria to concentrations at exposure points and calculates expected doses from plausible exposure scenarios.
Step 4:	Toxicity Assessment is comparable to Dose-Response Assessment. The section presents a toxicity profile and develops a dose response relationship for each Indicator Chemical. The general source for this information is the supporting literature for the standards and criteria for the constituents (even if a standard or criterion is not pertinent to the

exposure situation, the toxicity literature is often useful). Updated information will also be analyzed.

Step 5: Risk Characterization is titled the same in the Federal Register and the SPHEM. In this section expected doses are compared to the dose response estimation in order to quantitate risk for the site specific conditions.

The assessment of conditions at the Live Oak site is based on field observations as presented in the Remedial Investigation (RI) performed by Fishbeck, Thompson, Carr, and Huber, and Environmental Engineering and Management Ltd. Analytical data on a variety of samples taken by several organizations (Florida DER [FDER], EPA, P.E. LaMoreaux and Associates [PELA], Law Engineering and Testing Co. [LETCO], as well as Environmental Engineering and Management Ltd [EEM]) between 1982 and 1987 were assessed. Table 2-1 presents the chronology of sampling.

2.1 SELECTION OF INDICATOR CHEMICALS

All of the sampling rounds at the Live Oak Site have been focused on constituents potentially present, given the past use of the property as a wood preserving operation. Creosote is the principal preservative reported to have been used at the site during operations by Brown and Amax. Pentachlorophenol and arsenic-metal complexs (e.g. Chromated Copper Arsenate, Chromated Zinc Chloride) are also common wood preserving compounds. Although not reported to have been used by Brown or Amax, they have been addressed in sample analysis. The reasons for selection or rejection of a compound as an Indicator Chemical is detailed below and the final list of chosen constituents is given in Table 2-2.

TABLE 2-1
CHRONOLOGY OF ENVIRONMENTAL SAMPLING
LIVE OAK, FLORIDA

	Date	Sampled	n	For
FDER	Summer/82	surface and ground water (private wells)	4	"purgeable organics"
EPA	2/8/83	air		
EPA	2/8/83	ground water (private wells)	7	metals
EPA	2/9/83	surface water	3	creo. constituents, pcp
EPA	2/83	sediment (ditch and lagoon)	2	creo. constituents, pcp
EPA/PELA	6/83	borings		creo. constituents, pcp
PELA	6/24/83	surface water	3	creo. constituents, pcp
LETCO	9/83	borings	7	creo. constituents, pcp
EEM	9/84	surface soil (plant area)	7	creo. constituents, pcp
EEM	1/85	borings (ditch and plant)	10	creo. constituents, pcp
EEM	8/85	surface soil (plant)	26	creo. constituents, pcp
EEM	8/85	surface soil (wood storage)	16	creo. constituents, pcp
EEM	9/85	ground water (monitoring wells)	5	creo. constituents, pcp
EEM	10/86	ground water (monitoring well)	7	creo. constituents, pcp
EEM	1/87	ground water (monitoring well and private well)	3	creo. constituents

---

*creo. constituents = phenol, dibenzofuran and PAH associated with creosote.

*pcp = pentachlorophenol

TABLE 2-2
INDICATOR CHEMICALS

Compound	Weight of Evidence[a]	Solubility[b]	Log Kow[c]
Benzo (a) Anthracene	B2	14	5.61
Benzo (b) Fluoranthene	B2	0.8	6.06
Benzo (a) Pyrene	B2	3.8	6.06
Chrysene	B2	2	5.61
Dibenzo (a,h) Anthracene	B2	0.5	6.77
Indeno (1,2,3,cd) pyrene	C	0.2	
Fluoranthene	not ranked	260	4.90
Pentachlorophenol	D	14000	5.01

(a) EPA (1986). There are only a limited number of chemical compounds that have been demonstrated unequivocally to be human carcinogens. However, experimental and epidemiologic data are available that are suggestive of the carcinogenic activity of certain compounds. The EPA "weight-of-evidence" system for ranking from A to D (in decreasing order) the level of certainty that a compound is a human carcinogen is explained in the text. There are no A or B-1 level carcinogens present at the Live Oak Site. Certain PAH compounds present at the site have been rated at B-2 or C-level potential carcinogens. To obtain an appropriately conservative risk assessment the assumption is made that compounds are human carcinogens, even if there is limited certainty that this effect is real. As such, potential carcinogens down to level C weight of evidence have been selected. (EPA, 1986).

(b) Solubility (in ppb), at 25°C. Data for PAH from Craun and Middleton, 1984, data for pentachlorophenol from EPA, 1980c.

(c) Log octanol/water coefficient. Data for PAH from EPA, 1980b. Data for pentachlorphenol EPA, 1980c.

2.1.1 Creosote Constituents

Creosote is defined by its physical properties (selected specifically to make the material suitable for wood preservation) rather than its chemical composition. As a complex mixture, the toxicity of creosote may vary with each lot. Creosote is a mixture of aromatic compounds, including:

- Light aromatic compounds, including benzene, naphthalene and substituted aryl structures such as xylenes and toluene. These constituents are present at low levels because their boiling points are lower than the creosote fractionation temperature range.
- Phenol and substituted phenols (e.g. cresols).
- Polynuclear aromatic hydrocarbons (PAH).

Benzene and substituted benzenes have been detected once, by the FDER in 1982. At that time, they were found to be present in surface water of a shallow hole dug in the drainage ditch on site, but not in four ground water wells off site. The FDER and EPA (in summer of 1982 and February of 1983, respectively) did not find naphthalene, phenol or PAH in private ground water wells offsite (no constituents were found in the private well sampled by EEM in 1987), nor were they found in ground water monitoring well samples during the 1985 sampling rounds by EEM. No phenol was found by EEM in 1986 sampling of ground water, while very low concentrations of naphthalene and low molecular PAHs (acenaphthene, dibenzofuran, fluorene, phenanthrene and anthracene) were reported in two on-site monitoring wells in 1986 and 1987.

Phenol, naphthalene, and PAH were detected in surface water on site by FDER and EPA (dates mentioned above), but analyses by PELA in June of 1983 were negative.

Phenol was not detected to a significant extent in soils analysis during the Remedial Investigation, and was calculated to be present in air to the extent of about 0.5 ppb (EPA-Fit Study No. Z0830202, 1983), a concentration which is four orders of magnitude below the NIOSH Exposure Limit (NIOSH, 1985).

Thus, phenol has been eliminated from further consideration in the baseline risk assessment due to its limited presence at the site. Soil analyses done during the Remedial Investigation indicate that naphthalene, methylnaphthalene, and certain PAH are present in soils in specific areas at the site. However, naphthalene and methylnaphthalene have fairly low acute toxicity (lethal dose in humans is 2-15 g, Sandmeyer, 1981) and their chronic toxicity is inadequately studied. Therefore, the naphthalenes were not assessed further. PAH with an adequate toxicology base were selected as Indicator Chemicals.

The toxic effect of primary concern for PAH is carcinogenicity. The U.S. EPA has developed a "weight-of-evidence" system to classify the data that is suggestive of human carcinogenic activity of individual PAH compounds. The weight of evidence categories are:

1) A - human carcinogen. Demonstrated human carcinogenic activity.
2) B-1 - probable human carcinogen. Suggested by limited studies in humans.
3) B-2 - probable human carcinogen. Suggested by lifetime studies in animals.
4) C - possible human carcinogen. Suggested by limited studies in animals.
5) D - no data, or no demonstrated carcinogenic activity.

There are no A or B-1 level carcinogens among the PAH detected at the Live Oak Site. Six PAH compounds found at the site have EPA ratings of "probable" (B-2) to "possible" (C) human carcinogens (EPA, 1986) and have been chosen for detailed risk analysis in the present assessment. They are listed in Table 2-2.

Additionally, a non-carcinogenic PAH, fluoranthene, has been selected because there is adequate dose-response data to assess the toxicity of this compound (EPA, 1980a).

As will be detailed below, an acute dermal toxic effect of PAH may also be important at the Live Oak Site (An acute effect

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is one which may occur after a single instance of contact with the chemical, and generally happens within hours to days after exposure). All PAH will produce sun sensitivity to varying degrees. It is neither possible or necessary to determine the dermal toxicity risk of each PAH. Rather a value intended to protect against photosensitivity will be developed for total PAH. Even the PAH which have been eliminated from assessment for systemic toxicity will be included in this value.

2.1.2 Pentachlorophenol

Pentachlorophenol has been detected in surface water but not in ground water samples from the on-site or offsite monitoring wells. The compound appears to be less widely distributed in soils than creosote constituents. However there are concentrations in the sediments that could conceivably pose a risk through direct human contact or as a source for future ground water contamination. Thus, pentachlorophenol will be included as an Indicator Chemical in the quantitative risk assessment.

2.1.3 Metal-Arsenic Complexes

There are no reports of the use of metal-arsenic preservative at the Live Oak Site and analytic data does not indicate contamination with these compounds. The EPA (in February of 1983) detected copper in only one off-site well at 30 ppt, a value well below the Ambient Water Quality Criteria (1 ppm, EPA, 1980d) or proposed Recommended Maximum Contamination Level (1.3 ppm, FR 50 46968, November 13, 1985). Zinc was found in all wells sampled (at concentrations of 0.04 to 1.3 ppm) but adverse health effects from this compound have never been identified (FR 50 46981, November 13, 1985). PELA detected zinc and copper in the low ppb range (levels consistent with natural occurence, Bond & Straub, 1973) in lagoon water samples in June of 1983. At this time they found no arsenic or chromium. The limited distribution of metals and aresenic supports the claim that metal-arsenic preservatives were not used at the Live Oak Site. It is therefore not necessary to include these compounds among the Indicator Chemicals.

2.1.4 Summary

Selected Indicator Chemicals are listed in Table 2-2.

2.2 EXPOSURE ASSESSMENT

The Live Oak site is located west of the City of Live Oak, Suwannee County, Florida, a town of 6700 people (1980 population, Bureau of the Census, 1983) in the North Central portion of the state. There are private water supply wells in all directions from the site. The area surrounding the site is rural. A defunct sawmill operation and a construction company are located to the west and east of the site, respectively, but there are no permanent residents at these locations. The nearest permanent residents are located in a trailer court north of the site, and in new houses to the south. The site is posted with No Trespassing signs and locked cable gates have been installed across roadways. The property is partially fenced and, while the presence of unauthorized persons on site was documented in the Remedial Investigation, this is a rare occurrence due to the gates.

2.2.1 Exposure Pathways

At any site, humans may potentially be exposed to contaminants in air, water, or solid media (soils, sediments or sludges); directly, or via the food chain. The route of intake may be by ingestion, inhalation, or dermal absorption. Dermal contact even without absorbtion may also be pertinent for the indicator chemicals selected at the present site.

Air

Exposure to contaminants in air is not a likely exposure pathway for the Live Oak Site. The volatility of the indicator chemicals is generally low, and the nature of the waste precludes substantial entrainment. Finally, a majority of the local population lives at distances from the site that make air dispersion and dilution significant factors in diminishing exposure by this pathway.

Water

A significant potential exposure pathway off site is ingestion of water if the Floridan aquifer were to become contaminated as a result of leaching from the site. This pathway will receive substantial analysis in the following sections.

A pathway for direct contact with media onsite, which would include surface water is developed below, in the section on solid media.

Solid Media (soils, sediments, sludges)

A second pathway that may be significant for a subset of the population is the exposure to contaminants by direct contact with materials (sediments, soils and lagoon water) while on site. As the only authorized individuals on the property would be representatives of the PRPs or Agencies who are aware of appropriate protective measures for the location, the potentially at-risk population would be limited to trespassers.

Food Chain

Food chain exposure via plant crops is an unlikely pathway as surface run off from the site is captured by the lagoon or a swampy area to the west. The crops nearby have not required irrigation (FTCH, 1987, pg. 92). Wildlife species which may inhabit the site, and maybe hunted include raccoon, oppossum, and bobwhite. However, PAH and pentachlorophenol do not tend to accumulate appreciably in terrestrial animals, probably because the compounds are extensively metabolized and eliminated (EPA, 1980b). Thus, food chain exposure is eliminated as a major pathway for exposure.

2.2.2 Exposure Point Concentrations

The analysis of plausible exposure pathways presented above indicates that humans may potentially be exposed to Indicator Chemicals by drinking water or making direct contact with materials on the property. The following section determines what concentrations of Indicator Chemicals may exist at these exposure points now or in the future. The data will be utilized to determine potential human health impacts by comparing the values to relevant Standards and Criteria (see Section 2.2.3) and using the data for calculating human intakes and applying dose-response relations (see Section 2.3). The exposure point concentrations developed below are presented in Table 2-3.

Drinking Water Pathway

The EPA reported no detection of "extractable organics" in private wells during sampling in February of 1983. The new private well, sampled by EEM in January, 1987 had no detectable creosote constituents. The Remedial Investigation report indicated no indicator parameters in ground water samples taken in 1985 from monitoring wells at detection limits of 20 ppb for carcinogenic PAH compounds and 110 ppb for pentachlorophenol. In October 1986, fluoranthene and other low molecular weight PAH (acenaphthene, dibenzofuran, fluorene, phenanthrene, and anthracene) were detected in wells MW-4 and MW-8. Resampling

TABLE 2-3

CONCENTRATION OF INDICATOR CHEMICALS[a]
AT EXPOSURE POINTS
LIVE OAK, FLORIDA

Drinking Water Pathway

Indicator	Analytical Data (ppb) at MW-8	Analytical Data (ppb) Private Wells	Model (ppb) at MW-8	Model (ppb) 1600 ft. Well
Carcinogenic PAH	(20)[b]	d	.006	.0003
Acenaphthene	45/40	d		
Fluoranthene	2/2[c]	d	3	.1
Pentachlorophenol	(100)[b]	d		
Phenanthrene	8/11[c]	d	31	1

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a. All concentrations given in ppb (micrograms per liter). Analytic data for MW-8 is from October 1986 sampling, reported in the RI. Private well data is from EPA, 1983 sampling information. Acenaphthene and phenanthrene concentrations are given in addition to Indicator Chemicals. Acenaphthene was considered because it has an "organoleptic" AWQC. Phenanthrene concentrations were used to check the transport model values versus analytic data.

b. Not detected by REM in latest 1986 sampling round. Value given in parentheses is detection limit reported by laboratory.

c. Results of sampling in October, 1986 and January, 1987, respectively. These values were estimated by the laboratory. The normal detection limits are affected by the matrix, but generally are about 20 ppb.

d. Not detected by EPA, 1983, no detection limits given.

TABLE 2-3 (Continued)

Direct Contact Pathway

Indicator	Surface Water[a] Ditch	Surface Water[a] Lagoon	Surface Soils and[b] Accessible Sediments
Total PAH	329 ppb	76 ppb	12,357 ppm
Carcinogenic PAH	14 ppb	14 ppb	992 ppm
Fluoranthene	69 ppb	25 ppb	2,141 ppm
Pentachlorphenol	94 ppb	53 ppb	5,363 ppm

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a. Data from sampling round by NUS from ditch and lagoon (1 each) February 9, 1983, except Pentachlorophenol in lagoon, which is mean of sampling results from February 9, 1983 and June 24, 1983.

b. Mean concentration of 37 samples of surface soil and ditch sediments, as shown in Appendix B.

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in January, 1987 indicated again that low molecular weight PAH were present in MW-4 and MW-8, although the compounds were detected in lower concentrations than the previous sampling round.

In order to determine what future impacts might occur as a result of constituents at the Live Oak Site, a leachate and ground-water transport modeling effort was undertaken. The methods and results are detailed in an appendix to this report.

The PAH concentration inputs for the model were obtained from data on the sediments and soils in the lagoon area (PELA and EPA sampling of June 20-24, 1983) because this is the area of the largest waste load and the most likely place for ground water contamination to occur. Inadequate data were available to model possible leaching of pentachlorophenol, so this value is not reported.

The model utilized for determining the potential magnitude of leaching from the lagoon material (the EPA Organic Leaching Model, or OLM), proved valid only for soils beneath the sludge layer in the lagoon. For sludge contaminants, the model consistently predicted leachate concentrations in excess of the solubility limits of the carcinogenic PAH. Thus, the solubility limits were used as a sludge source term for transport models. Transport in the unsaturated zone was calculated by a one-dimensional analytical model. This model provided mass fluxes as a source term to the saturated zone model (the HPS model), which was developed by ERT. These models (described in detail in Appendix A) were utilized to predict potential drinking water impact of the lagoon in its present condition.

The primary point of impact chosen was the nearest downgradient well from the source. This is Well Number 15 on sheet 10 in the Remedial Investigation report, which is approximately 160C feet from the source. Although an actual well was chosen, the models make the assumption that the point is directly down gradient. Thus, the values predicted from the models will be generally valid for 1600 feet down gradient.

regardless of direction of ground water flow. Additionally, solutions to the transport models were calculated using MW-8 as an exposure point. This activity served a dual purpose. As some materials were detected in MW-8 during the 1986 sampling round, it was possible to compare model output to actual data in order to ensure that the model was conservative (the only Indicator Chemical found in this well was fluoranthene; transport of an additional low molecular weight PAH, phenanthrene, was modeled to provide an additional comparison to analytical data). Additionally, MW-8 is only a few feet from the lagoon, the area of highest waste load and potential for leaching to ground water. It therefore may serve as a point for making conservative, possibly "worst-case", predictions of potential ground water contamination. Predicted concentrations are given in Table 2-3.

Direct Contact Pathway

Of concern for direct contact are those soils, sediments and surface waters to which project personnel or trespassers may have access. For the analysis, all surface water, the ditch sediments, and surface soils were considered to be accessible. The highest value and the mean concentration of the indicator chemicals in each of the accessible media is given in Table 2-3.

2.2.3 Comparison of Exposure Point Concentrations to Standards and Criteria

Drinking Water Pathway

There are few criteria and no standards which are relevant to and applicable to the exposure pathways at the Live Oak site. The Ambient Water Quality Criteria (AWQC), which were intended for use with surface water, may be used to predict health risk from drinking water. These criteria are intended to minimize health risk from ingestion of water as well as ingestion of aquatic species whose flesh may contain contaminants partitioned from the water. By eliminating the allowance for ingestion of aquatic biota, AWQC values may be applied to aquifer water, where only the drinking exposure occurs. The adjusted AWQC, taken from the Superfund Public Health Evaluation Manual (pages 61-64) are given in Table 2-4.

An AWQC for a low molecular weight PAH, acenaphthene, is also given in Table 2-4. This is the only non-Indicator Chemical detected in the on-site wells for which an AWQC exists. The AWQC for acenaphthene is not designed for protection of health (there is little data on the health effects of acenaphthene, which is why it was not chosen as an Indicator Chemical). Rather it is an "organoleptic" limit, indicating a concentration at which water might be impacted relative to taste or smell.

The only other relevant value is a proposed Maximum Contaminant Level Goal for pentachlorophenol under the Safe Drinking Water Act, which is also given in Table 2-4.

Chemical analysis of samples from private wells (EPA sampling of February, 1983) revealed no Indicator Chemicals. This would indicate that the potential human health impact of indicator chemicals either on-site or off-site, via the drinking water pathway is low. However, the EPA data is difficult to interpret because detection limits were not provided. Further, the AWQC for carcinogenic PAH is an extremely small concentration and analytic methods with detection limits in the range of the criterion are not practically feasible. Thus, the detection limits for carcinogenic PAH reported in the Remedial Investigation are above the AWQC. It is therefore not possible to use the method of comparison to criteria to determine the extent of public health impact of carcinogenic PAH using either the EPA or the Remedial Investigation data. If it is assumed that the EPA achieved detection limits comparable to those reported for

TABLE 2-4

STANDARDS AND CRITERIA FOR INDICATOR CHEMICALS

Compound	Ambient Water Quality Criteria[a]	Recommended Maximum Contaminant Level [b]
Carcinogenic PAH	0.031 ppb[c]	
Acenaphthene	20 ppb[d]	
Fluoranthene	188 ppb	
Pentachlorophenol	1010 ppb	200 ppb

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a. AWQC has been adjusted for ingestion of water only. All values given in units of ppb (micrograms per liter).

b. The value is a proposed RMCL (50 FR 47002, November 13, 1985).

c. EPA has calculated cancer risks for various concentrations. The value for a 1 chance in 100,000 risk is given here.

d. The actual AWQC for acenapthene is indicative only of an "organoleptic" level at which some imapct on water taste or smell might be affected. No health impact is expected at this level.

analyses in the RI for fluoranthene, pentachlorphenol, and acenaphthene (20 ppb, 100 ppb, and 20 ppb, respectively), no impact on human health or welfare would be expected, based on the method of comparison to criteria.

In the Remedial Investigation study, fluoranthene was estimated to be present in well MW-8 at a concentration of 2 ppb in October, 1986 and 11 ppb in January, 1987; and pentachlorophenol was not detected (detection limits, 100 ppb October, 1986). These concentration values are substantially below the AWQC listed in Table 2-4. Provided that the monitoring wells provide a generalized picture of ground water quality at the Brown Wood Treating Site, no impact on public health is indicated by the method of comparison to criteria for these Indicator Chemicals. The October, 1986 sampling indicated 45 ppb acenaphthene is present in MW-8. This does not indicate any health impact, but does suggest the possibility that the odor or taste quality of water could be impacted at this "worst-case" location.

Data from the ground water modeling study predict higher concentrations of fluoranthene and phenanthrene in MW-8 than have been detected. The difference in values may be due to conservatism of the model. Therefore, the predicted concentrations appear to be conservative values for estimating the current impact of Indicator Chemicals potentially leaching to ground water. Model output for total carcinogenic PAH and fluoranthene at the 1600-foot well are 0.0003 and 0.1 ppb, respectively (pentachlorophenol values could not be calculated). Concentrations predicted for on site location, MW-8 are 0.006 ppb and 3 ppb for carcinogenic PAH and fluoranthene, respectively. The carcinogenic PAH values in each of these locations are below the concentrations set in the Ambient Water Quality Criteria Documents as values where carcinogenic risk would be relatively low. The predicted fluoranthene concentration at the off-site and on-site well locations are substantially below the criterion. Thus, by the method of comparison of modeling results to criteria, impact of the Indicator Chemicals from the site appears very low.

Direct Contact Pathway

Standards or Criteria for allowable concentrations in soils, sediments, or lagoon water are not available. The significance of contamination in these media have been assessed using standard risk analysis procedures.

2.2.4 Estimation of Chemical Intakes

Chemical intakes have been calculated with the aid of exposure scenarios relevant to the pathways identified above. Plausible mechanisms by which intake may occur has been outlined and an estimate of the magnitude of the intake has been calculated from standard values for human activities leading to the exposure (e.g. volume of daily fluid intake). Intake values have been converted to units of micrograms of Indicator Chemical per kilogram of body weight per day, to make them compatible with the dose-response relations developed in the subsequent Section.

Drinking Water Pathway

Most of the population of Live Oak consumes water from wells in the Floridan aquifer. Although the general flow of ground water in the area of the site is away from populated areas, the possibility has been considered that exposure to constituents could occur if ground water were contaminated by materials from the Live Oak Site. To calculate the magnitude of exposure (in micrograms of constituent per kilogram body weight per day) one multiplies the amount of drinking water consumed daily (assumed to be 2 liters per day in adults and 1 liter per day in children, EPA, 1986) by the concentration of Indicator Chemicals predicted at the exposure point and corrects for body weight (assumed to be 10 kg for small children and 70 kg for adults):

$$\text{Water Ingestion, Daily dose } (\mu g/kg\ day) = \frac{C_w \times WC}{BW}$$

where,

C_w = Concentration of constituent in water (µg/L)
WC = Water Consumption (1 L/day for children; 2L/day for adults)
BW = Body Weight (10 kg for children, 70 kg for adults)

No Indicator Chemicals have been detected in ground water at the off-site exposure points. For the on-site location, only fluoranthene was detected in analytical samples. Thus, only model predictions for carcinogenic PAH (as shown in Table 2-3) were used. In the case of fluoranthene, model predictions were used for the off-site location, and both analytic results and model predictions were used for on-site assessment. This is equivalent to assessing the health risk now and in the future, given the time projection of the transport model. Because neither analytic data nor model prediction data were available for pentachlorophenol, the intake (daily dose) equation was applied to the detection limits of the RI study. The values represent a "worst case" for intake because the actual concentrations are below the detection limit, if they are present at all. For the off-site location, the intake prediction is even more conservative, because dilution of pentachlorophenol would occur between the entry point into the aquifer at the site and ultimate exposure points.

Both child and adult intake values were calculated for pentachlorophenol and fluoranthene, because it is presumed that toxicity could appear after a relatively short period of exposure. In contrast, the value that must be applied to dose response relations for carcinogenic toxicants is a lifetime daily dose. Because most of a lifetime is lived as an adult, only an adult intake was calculated for carcinogenic PAH. The calculated values are presented in Table 2-5.

TABLE 2-5

PREDICTED INTAKES OF INDICATOR CHEMICALS

DRINKING WATER PATHWAY

LIVE OAK, FLORIDA

Compound	On-Site (µg/kg day)[a,b] Child Intake	Adult Intake
Carcinogenic PAH	-------	0.0002
Fluoranthene	0.2 - 0.3	0.6 - 0.09
Pentachlorophenol	<10	<2.86

Compound	Off-Site (µg//kg day)[b,c] Child Intake	Adult Intake
Carcinogenic PAH	-------	0.0000086
Fluoranthene	0.01	0.0029
Pentachlorophenol	<10	<2.86

---

a. For predicted or measured concentrations in MW-8. Analyses were below detection limits for PAH and pentachlorophenol. Values have been calculated from model predictions for PAH and detection limits for pentachlorophenol, the latter is noted as being "less than (<)" the intake predicted from contamination at the detection limit. Range of values calculated for fluoranthene are from detected concentration to concentration predicted by the model. All values in units of micrograms Indicator Chemical per kilogram body weight per day (µg/kg day).

b. Because toxic effects for Carcinogenic PAH require lifetime dosing, no child's dose was calculated.

c. For predicted concentrations at the 1600 foot well. Model predictions used for carcinogenic PAH and fluoranthene. Detection limit used for pentachlorphenol. All values in units of micrograms Indicator Chemical per kilogram body weight per day.

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TABLE 2-5 (Continued)

Predicted Intakes of Indicator Chemicals
Direct Contact Pathway[a]
Live Oak, Florida

	μg/kg day
Carcinogenic PAH	0.0076
Fluoranthene	0.58
Pentachlorophenol	0.23

---

a. Intake of materials by children visiting the site once monthly and consuming 55 mg soil per visit for five years. All values in units of micrograms constituent per kilogram body weight per day (μg/kg day). Values are lifetime average daily doses for carcinogenic PAH and average daily doses for fluoranthene and pentachlorophenol.

Direct Contact Pathway

Because the site is not completely secured, it is possible that people might trespass and make direct contact with contaminated media onsite. There are two aspects to such an exposure. People might experience a systemic exposure as the result of inadvertent ingestion of materials clinging to hands or articles which may be placed in the mouth. Contaminated soils and ditch and lagoon sediments (those not covered continually by water) are the media of concern for this scenario. People might also experience a dermal effect as a result of contact with contaminated materials. Soils, sediments and surface water are all of concern for this scenario.

Ingestion of materials is normally considered to be of concern for children only. It has recently been suggested that a child between the ages of two and six years may be assumed to ingest 55 mg of soil per day while outdoors (Clausius, et al. 1987). The question for a rural site such as the Live Oak property is whether children of this age would ever reach the site and, if so, how often. It is unlikely that frequent visits by this age group would occur in the present condition of the site. For the purposes of quantitation, intake predictions will be made for children visiting the site once monthly from age two through six years. This seems conservative, given the distance of the site from residences, the limited access to the property, and the fact that the site is not on the route to a school, playground or other destination attractive to children. Unknown future uses of the property, unless otherwise restricted, might include more frequent presence of children. Notice that because a lifetime average daily dose is required for ascertaining chronic risks of carcinogenic PAH, an adjustment must be made for the proportion of a 70 year lifetime in which exposure occurs. This correction is not applied for fluoranthene or pentachlorophenol, where toxicity may be expressed after shorter periods of dosing. Children in this age group are assumed to have an average body weight of 17 kg (EPA, 1986). Expressed mathematically the intake would be:

$$\text{Soil ingestion daily intake } (\mu g/kg \text{ day}) = \frac{C_s \times SC}{BW} \times f \times d$$

where,

C_s = Constituent concentration in soil (µg/kg)
SC = Soil consumption (0.000055 kg/day)
BW = Body weight (17 kg)
f = Frequency of exposure (1 day/30 days)
d = Duration of exposure (used for carcinogens only, to obtain a lifetime average daily intake; 5 years/70 years)

This formula has been applied to the mean soil concentration data to model the potential impact of an individual taking a "random walk" around the site and picking up contaminated materials from several places. The results are presented in Table 2-5.

A more plausible scenario for health impacts from direct contact with contaminated material at the Live Oak Site is toxic effects on the skin. This scenario is more plausible because it would affect adults as well as children, and children are less likely to be on site. Further, this is an acute effect of the Indicator Chemicals. Thus, it is not a function of frequency of contact. It is not really necessary to calculate an "intake" for this type of topical effect. In Section 2.3.4 concentrations of PAH (total) and pentachlorophenol that might be associated with dermal effects are developed.

2.3 TOXICITY ASSESSMENT

2.3.1 Carcinogenic PAH

PAH are formed as a result of elevated temperature processes such as fires, petroleum-synthetic mechanisms in the deep subsurface, and anthropogenic activities such as operation of internal combustion engines and incineration or other combustion of refuse, forest, and agricultural products. The largest contribution of PAH to the environment are the man-made combustion sources mentioned above. PAH are not generally intentionally synthesized, but are obtained by refining natural materials for use as fuels, lubricants, preservatives and starting materials for petrochemical manufacture.

Only certain PAH have been identified as having the potential to cause cancer. Such carcinogenic PAH generally tend to be high in molecular weight, have at least 3 aromatic rings (usually more), have low water solubility, are easily absorbed by humans, and have very low acute toxicity. PAH have not been unequivocally demonstrated to be carcinogenic in humans. However, by extrapolation from health effects in individuals who smoke, and from animal data on certain PAH compounds there is reason to believe that some PAH are carcinogenic in humans. The U.S. EPA "weight-of-evidence" system to classify carcinogen data has been described previously (Section 2.1.1). There are no A or B-1 level carcinogens among the PAH detected at the Live Oak Site. Six PAH compounds found at site have EPA ratings of "probable" (B-2) to "possible" (C) human carcinogens (EPA, 1986) and have been chosen for detailed risk analysis in the present assessment.

In determining one set of criteria, the AWQC, EPA (1980a) used animal dose-response data for benzo[a] pyrene to establish a criterion for all carcinogenic PAH (summed quantities). This approach is very conservative, because the carcinogenic potency of benzo[a]pyrene is probably greater than other PAH.

The approach is also an oversimplification because the potency of an individual PAH may change according to the route of exposure and the presence of other compounds in the exposure mixture. Applying dose-response data from benzo[a]pyrene to other PAH is, nonetheless, the only method currently more available.

Studies on chemical carcinogenesis suggest that, for some compounds, no threshold for the effect exists. That is, certain carcinogens, even in extremely small doses, will pose some risk of cancer. This assumption is incorporated into the cancer dose-response assessment for PAH. Neal and Rigdon (1967) gave mice feed containing between 1 and 250 milligrams per kilogram (ppm) benzo[a]pyrene and found that more treated animals developed stomach tumors than the control group. The increased tumor incidence was dose dependent. After adjusting the doses to correct for presumed differences in mouse versus human metabolism, this data was used by the EPA Carcinogen Assessment Group in a computer program which calculates the upper 95 percent confidence interval on the slope of a dose response line fitted to an equation modeling the assumed no threshold, multistage mechanism of chemical carcinogenesis. The value, called a "potency slope," is 0.0115 per microgram per kilogram body weight per day for ingestion exposures to benzo[a]pyrene. The potency slope indicates that an individual consuming 1 microgram benzo[a]pyrene per kilogram body weight, daily, for life, might have a risk of contracting cancer of about 1 chance in 100 over that of the non-exposed individual (note that this is an upper bound on the estimate, the actual risk is more likely to be lower). Because the dose-response relation is presumed to be linear, multiplying the predicted lifetime daily intake of carcinogenic PAH by the potency slope will give an upper bound estimate of excess cancer risk from exposure to constituents at the Live Oak Site (by the routes previously outlined).

2.3.2 Fluoranthene

Fluoranthene is among the PAH compounds which has been demonstrated to lack carcinogenic activity both in skin painting tests and by subcutaneous injection. Fluoranthene has also been shown to lack mutagenic activity in the Ames test (mutagenic activity is often related to carcinogenicity). Thus, risk analysis for fluoranthene will be conducted on other potential toxicities.

The acute toxicity of fluoranthene appears to be low (LD_{50} in rats 2g/kg: Smyth, et al. 1962 as quoted in EPA, 1980a). The AWQC for fluoranthene (EPA, 1980a) was calculated on the basis of an extremely limited study by Hoffman, et al. (1978). It is questionable whether the data are truly sufficient to establish a criterion. The data are applied to this risk analysis with skepticism. Hoffman, et al. applied 50 microliters of a 1% solution of fluoranthene to the backs of mice 3 times weekly for one year and saw no mortality in the animals (for up to 15 months). If one presumes that the entire amount of fluoranthene was absorbed and that the average weight of a mouse is 35 g, one may calculate a "no-effect level" of 6.12 mg/kg body weight. Presumably this type of toxic effect has a threshold. That is, there exists a dose below which no individual will respond with a toxic effect. It is common practice to apply a "safety factor" to an experimentally determined no-effect level to provide reasonable certainty that a sub-threshold for toxic effects is obtained. Often the no effect level is divided by 10 to allow for possible differences in sensitivity between man and animals and another factor of 10 to correct for possible differences in sensitivity among humans. In the case of fluoranthene, an additional 10 fold safety factor was applied by the EPA due to the small amount of data available in the study. Thus, the corrected no effect level, called an "Acceptable Daily Intake" (ADI) for fluoranthene was determined to be 6.12 μg/kg day. EPA calculations of fluoranthene burdens from other sources

(.016 mg/man day from diet and .0001 mg/man day from the air) do not appreciably change this value. The SPHEM gives no acceptable intake value for fluoranthene. Thus, the ADI developed in the AWQC will be used for assessing risk from fluoranthene at the Live Oak Site. Applying the threshold concept, if the exposure is below the ADI, no risk would be expected. If exposure is above the ADI risk may be present. This risk is not quantifiable, but it may be qualitatively stated that the greater the exceedance of the ADI, the more likely it is that a toxic effect will be seen.

2.3.3 Pentachlorophenol (PCP)

The sole use of PCP is as an antimicrobial preservative of wood products. PCP is well absorbed by the dermal, inhalation, and ingestion routes of exposure. Acute toxic episodes in humans have been reported after dermal and inhalation exposures (EPA, 1985b). The acute toxic effects of sweating, fever, and rapid heart rate are probably related to the ability of PCP to interrupt energy metabolism. Other acute toxic effects of PCP are related to the irritative properties of the compound and include reddening and painful sensations of skin immersed in the compound, irritation of the throat after drinking contaminated water (12.5 milligrams per liter), and congestion of eyes and nasal passages. Effects reported in humans with possible chronic exposure to PCP may include liver, kidney, bone marrow damage, and infections which may be related to poor immune function.

To establish a dose-response relationship for the systemic effects of PCP, the EPA has relied on a study conducted by Schwetz, et al. (1978), which reports on chronic toxic effects and the reproductive ability of rats. Schwetz, et al. noted that in the reproductive study when animals were fed 0, 3 or 30 milligrams per kilogram body weight (ppm) per day of PCP prior to and during the gestation period, animals in the highest dose group only had a lower percentage of live births than controls. Each dose group contained 10 males and 20 females. The offspring of the high dose mothers were lower in weight and survived less often than untreated animals. This would make the lower dose (3 milligrams per kilogram (ppm) per day) a "no effect" level.

The two year chronic study contained 5 different dose levels (0, 1, 3, 10, 30 milligrams PCP per kilogram per day). Each dose group contained 25 rats of each sex with 2 additional rats per group maintained for tissue specimens used for chemical analysis. The no-observable-adverse-effect-level (NOAEL) for PCP, based on chronic toxicity, was 10 milligrams per kilogram per day among males and 3 milligrams per kilogram per day among females.

The EPA had used the standard practice of applying a 100 fold safety factor to the NOAEL dose to arrive at an acceptable intake, subchronic (AIS) of 0.03 milligrams per kilogram body weight per day. No further uncertainty factors were applied to the AIS to derive the acceptable daily intake, chronic (AIC): it is also 0.03 milligrams per kilogram body weight per day. Presumably toxic effects such as those observed in the PCP experiments here have a threshold. That is, there exists a dose below which no individual will respond with a toxic effect. The purpose of the safety factor (which is a factor of 10 to allow for possible differences in sensitivity between man and animals and another factor of 10 to correct for possible differences in sensitivity among humans) is to provide reasonable certainty that the AIC will be below the threshold for toxic effects. The AIC derived by the EPA will be used for the present assessment of PCP impact at the Site. If predicted intake is below this value, no risk of toxic effect is expected. If intake is greater than the AIC, a toxic effect may occur. The larger the intake value is over the AIC, the greater likelihood of a toxic effect.

2.3.4 Noncarcinogenic Dermal Toxicity of Indicator Chemicals

The values presented above are derived from studies of the systemic toxicity of Indicator Chemicals. As has been suggested in previous sections of this report, it is possible that the most likely exposure for persons on site at the Live Oak Site may be brief dermal contact with contaminated soils. Such an exposure might not result in significant systemic absorption of compounds or might not be of sufficient frequency for a chronic systemic effect to occur. However, it could result in dermal irritation or other effects. Some suggested concentration values, that may result in these effects, are derived from the data presented below.

Creosote Constituents

A number of papers have noted that various PAH compounds, when applied to human skin, will produce an enhanced sunburn reaction on exposure to ultraviolet light. The phototoxicity effect is usually reversible, but could cause transient problems for workers or trespassers at the Site. In 1980, an exchange of correspondence between Urbanek and Walter in the Journal of the American Academy of Dermatology indicated that 0.25 percent anthracene dissolved in petroleum did not produce phototoxicity but did cause a stinging sensation and transient hive-like reaction in a small number of patients while 0.1 percent solution was without any toxic effect. For the purpose of the current assessment, 0.1 percent (1,000 ppm) will be considered a no-effect level for all PAH. This value may be overly conservative in that the availability of PAH adsorbed to soil particles may not be as great as that in a homogeneous solution in medicinal preparations. The value may be a poor estimate toxicity in that PAH other than anthracene are present in soil at the Live Oak site. The other PAH have not been studies, but may be more or less photoreactive than anthracene alone. It is notable, however, that a 1 percent (10,000 ppm) coal tar solution was only minimally effective in producing a phototoxic reaction (Tannenbaum, 1975). This preparation would be expected to contain a variety of PAH compounds.

Pentachlorophenol

Deichman, et al (1942) reported on the effects of PCP administered dermally in animals. In this study, application of 10 ml of 1 percent PCP in mineral oil (10,000 ppm) for 4 hours was without local dermal effect in 21 days of dosing. Solutions of 5-10 percent gave positive or negative dermal results depending on the volume of material applied and the vehicle in which the PCP was dissolved. Using a 10 fold safety factor for animal to human extrapolation on the 10,000 milligrams per kilogram (ppm) no effect level for dermal irritation would indicate an acceptable concentration for protection from this effect would be 1,000 milligrams per kilogram (ppm). This value may be overly conservative in that the availability of PCP adsorbed to a soil or sludge particle may not be as great as that in a homogeneous solution in organic solvents.

2.4 RISK CHARACTERIZATION

Table 2-6 presents numerical estimates of carcinogenic risk and other toxic effects for the scenarios developed in this report.

It is not possible to calculate a drinking water cancer risk from actual data because of the lack of detection limit information for the sampling round on private wells. The modeling effort for the potential future condition of the aquifer if no action is taken at the site indicates that a cancer risk of slightly less than 1 chance in 10,000,000 could be incurred by drinking water from the aquifer if leaching

TABLE 2-6
HEALTH RISKS FROM INDICATOR CHEMICALS FOR PRESENT CONDITION OF SITE
LIVE OAK, FLORIDA

Carcinogenic PAH[a]

Source	Risk
Ingestion of Drinking Water	
On Site	2.3 chances in 1,000,000
Off Site	9.9 chances in 100,000,000
Ingestion of PAH in Surface Soils	8.8 chances in 100,000

---

a. Calculated for the sum of carcinogenic PAH, assuming all have equivalent potency as benzo(a)pyrene (potency slope = .0115/ug/kg day).

TABLE 2-6 (Continued)

HEALTH RISKS FROM INDICATOR CHEMICALS
FOR PRESENT CONDITION OF SITE
LIVE OAK, FLORIDA

Health Impacts of Fluoranthene

Source	Predicted Intake/ADI (a)	
Ingestion of Drinking Water	Child	Adult
on site	0.03-0.05	0.01-0.015
off site	0.002	0.0005
Ingestion of Fluoranthene in Surface Soils	0.09	

a. A value of less than one indicates intake is below ADI (6.12 μg/kg day) and virtually no risk is likely. Values above one indicate possible risk of toxic effect.

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TABLE 2-6 (Continued)

HEALTH RISKS FROM INDICATOR CHEMICALS
FOR PRESENT CONDITION OF SITE
LIVE OAK, FLORIDA

Health Impacts of Pentachlorophenol

Source	Predicted Intake/ADI (a)	
Ingestion of Drinking Water	Child	Adult
on site	<.33	<0.1
off site	<.33	<0.1
Ingestion of Pentachlorophenol in Surface Soils	0.008	

a. A value of less than one indicates intake is below ADI (30 µg/kg day) and virtually no risk is likely. Values above one indicate possible risk of toxic effect.

occurs. For the onsite water location, the risk is approximately 2 chances in 1,000,000. It should be pointed out that this risk estimate is an upper bound for the following reasons:

- The potency slope value used to estimate risk is the 95% upper confidence bound on the dose response relation. Thus, the actual risk is likely to be lower.
- The concentrations are predicted for the closest existing off site well and a monitoring well extremely close to a potential source of contamination.

Cancer risks from direct contact (ingestion) with contaminated soils onsite are greater in magnitude than the drinking water scenario at slightly less than 1 chance in 10,000, but also less likely to occur. The location and characteristics of the site preclude regular visits by small children. Although this scenario does not represent a probable situation for evaluating the current impact of the site, it should be considered when planning future uses of the property.

No exposure scenario revealed a health impact from fluoranthene. All predicted intakes were less than the ADI, indicating virtually no risk of a toxic effect from this compound.

Ground water analysis indicated pentachlorophenol was undetectable at a limit of about 100 ppb. Even if pentachlorophenol were present at the level of detection, no health risk would be expected as ingestion of this level pentachlorophenol would provide a dose that is still below the acceptable daily intake.

A possible risk not quantified in the Tables is the potential for dermal effects from direct contact with surface materials on site. As with other scenarios, it is difficult to quantify the likelihood that an individual would be on site and make contact with contaminated soils, sediments or surface water. However, because dermal toxicity is an acute effect, requiring only a single visit to the site, one would intuitively rate this as a more likely scenario than those which require regular presence on the property. In the Toxicity Assessment section it was conservatively estimated that 1,000 ppm of total PAH could cause photosensitization and 1,000 ppm pentachlorophenol might cause dermal irritation. There are surface sediment and soil locations where these values are exceeded. However, surface water concentrations are substantially below this concentration. Thus, dermal effects from contact with solid media seem plausible, but surface water does not present this hazard.

3. DEVELOPMENT OF PERFORMANCE GOALS

In compliance with the Superfund Public Health Evaluation Manual guidance on source control remedial alternatives, an analysis determining "acceptable" residual levels of chemicals is presented here.

No evaluation of fluoranthene and pentachlorophenol were done as the baseline assessment indicated no present or future health impacts of the compounds. The low level of carcinogenic risk for the drinking water scenario also precluded the necessity of performance goals based on this limited health impact.

It was determined in Chapter 2 that the most likely health risk of the Live Oak Site was the potential for non-carcinogenic, acute dermal effects produced by direct contact with PAH and PCP in surface soils and sediments. Thus, a concentration limit should be put on PAH and PCP in soils with which people might make direct contact. Total PAH should not exceed 1000 ppm in surface soils in order to avoid possible phototoxic reactions. Pentachlorophenol should not exceed 1000 ppm to avoid dermal irritation. As detailed in the Feasibility Study Report, the preferred remedial alternative has been designed to meet this performance goal. All materials in the lagoon, ditch, and soil containing concentrations in excess of 1000 ppm PAH or PCP will be removed.

A calculation may also be made for the risk reduction achieved by the preferred alternative for the soil ingestion scenario, although it is a less likely exposure, given the current use of the site. In Section 2.4, the cancer risk associated with the mean concentration of surface soil at the site was estimated for a scenario in which children ingested soil at the site once a month for 5 years. Because the risk estimate was related to soil concentration in a linear manner, the risk reduction associated with the decreased average soil concentration at the site may be easily calculated using the equation developed on page 2-22 and the potency slope (0.0115/mg/kg day). If one remediated soil containing greater than 1000 ppm creosote substances in order to limit the risk of transient dermal effects, the average across the site of carcinogenic PAH concentrations remaining in the soil would be approximately 14 ppm. This value was calculated by substituting the concentration of carcinogenic PAH in the next strata sampled (if available) for each sampling point where values of PAH or PCP were in excess of 1000 ppm. The calculations are presented in Appendix B.

As shown in Table 3-1, 14 ppm of carcinogenic PAH would be associated with a substantially reduced cancer risk due to ingestion of soil: 1.2 chances in 1,000,000.

TABLE 3-1
COMPARISON OF HEALTH RISKS
BEFORE AND AFTER
PREFERRED REMEDIAL ALTERNATIVE
LIVE OAK, FLORIDA

	PAH (a)		PCP (b)		Fluoranthene (b)	
	Before	**After**	**Before**	**After**	**Before**	**After**
Acute Direct Contact	possible	unlikely	possible	unlikely	included in total PAH	
Soil Ingestion	8.8	0.12	0.008	NC (c)	0.09	NC (c)
Drinking Water, Off-Site	0.099	NC (c)	<0.1-<0.33	NC (c)	0.002-0.0005	NC (c)
Drinking Water, On-Site	0.23	NC (c)	<0.1-<0.33	NC (c)	0.03-0.015	NC (c)

(a) PAH risk is calculated as cancer risk per 100,000 chances, except in the case of acute direct contact, where the likelihood of dermal photosensitization is estimated qualitatively.

(b) PCP and Fluoranthene risk is calculated as the proportion of the AIC represented by the estimated intake except in the case of acute direct contact, where the likelihood of dermal irritation is estimated qualitatively. As noted, fluoranthene is among the total PAH assumed to have photosensitizing properties and is included under the PAH category.

(c) Where small risk was estimated for the current condition of the site, risk values for the remediated site were not calculated (NC).

4. SHORT-TERM RISKS OF REMEDIAL ALTERNATIVES

Other than the No Action alternative, the remedial actions chosen for screening for the Live Oak site are source control measures. The alternatives are off-site disposal, off-site incineration, on-site incineration, solvent washing techniques, and combinations of these techniques. Short-term health risks from these activities are discussed in a qualitative manner below.

4.1 Dermal Toxicity

The possibility that individuals might make contact with materials at the Site has been covered in the baseline assessment. It was stated that site access was difficult and it was unlikely that individuals would be present at the site with any frequency. When remedial measures are implemented, the number and frequency of personnel on the site will increase. This may increase the short-term risk due to direct contact with contaminated materials. However, it should be recognized that an approved Health and Safety Plan will be implemented. That plan will provide for minimizing prolonged contact with media containing more than 1000 ppm pentachlorophenol or 1000 ppm creosote substances and frequent cleansing of unprotected skin.

4.2 Transportation Risks

Off-site remedial activities involving transportation (e.g., off-site disposal, off-site incineration, transportation of recovered material) may be associated with vehicular accidents. If vehicles involved in the remedial action are subject to the same risks as Florida motorists in general, statistics would indicate that 3.3 traffic deaths will occur for every 100,000,000 miles driven (Land, et al., 1985). The mileage estimated from remedial actions (maximum, about 350,000 miles) would be associated with a population risk of accidental death of about 1 chance in 100 (n.b this is a population risk which cannot be compared to the individual risks derived for carcinogens previously - if a comparison is pertinent, the transportation risk would be roughly equivalent to the population cancer risk of the City of Live Oak (6700 residents) if the average individual risk was 2 in 1,000,000).

A second aspect of vehicle accident risk is the human health impact of dispersion of contaminated materials. A contingency plan will address this possibility if off-site remedial actions are chosen.

4.3 Air Emissions

If incineration alternatives are chosen, there is a possibility that emissions from this technology could have a health impact. Adequate remediation design is required to minimize this risk.

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APPENDIX A
GROUND WATER
CONTAMINANT MODELING

APPENDIX: GROUND WATER CONTAMINANT MODELING

A.1 Modeling Techniques and Assumptions

A.1.1 Leachate Model

A model was used to attempt to determine concentrations of contaminants in leachate. The EPA OLM (Organic Leachate Model) model was developed for delisting evaluations by statistically fitting actual leachate and waste mass concentrations (EPA 1986). The model is expressed mathematically as:

$$C_L = 0.00211\ C^{0.678}\ S^{0.373} \qquad \text{(A-1)}$$

Where;

CL = contaminant concentration in leachate
C = contaminant concentration in waste
S = solubility of contaminant

As will be seen in section A-2, the OLM model proved invalid for some of the contaminants at the Live Oak site, for which it was replaced by the conservative assumption that these contaminants were present in leachate at their solubility limit.

A.1.2 Ground-Water Models

A.1.2.1 Unsaturated Zone

An analytical solution (Battelle, 1986) was used to calculate the moisture content as a function of steady state flux in the unsaturated zone between the lagoon and the limestone aquifer. The unsaturated zone moisture content, θ, was used in calculations to estimate the retardation of the contaminants as they passed through the unsaturated zone.

The following expression was derived from Darcy's equation for steady state flow and soil characteristic relationships described by Campbell (1974).

$$\theta = \left(\frac{q}{Ksat}\right)^{m}\theta s \qquad \text{(A-2)}$$

Where:

θ = the unsaturated moisture content
q = the steady state flux (cm./min.)
Ksat = saturated hydraulic conductivity (cm./min.)
θs = saturated moisture content
m = 1/(2b + 3) where b is negative one times the slope of the log-log plot of the matric potential, ψm, versus θ.

The equation is based on the following assumptions.

- flow is one-dimensional in the vertical direction
- water flow is steady state
- water table conditions exist at the lower boundary
- the upper boundary condition is constant flux
- soil characteristics (θ versus ψm and hydraulic conductivity versus ψm) are constant with depth and
- the hydraulic gradient is vertically down and equals unity (drainage is due strictly to gravity and capillary forces are dominated by gravitational forces).

The moisture content in the unsaturated zone, θ, was used in the calculation of the retardation factor, R, for each constituent from the following equation.

$$R = 1 + \frac{\rho(Koc)(foc)}{\theta} \quad \text{(Yaron et al 1984)} \qquad \text{(A-3)}$$

Where:

Koc = constituent-specific organic-carbon partition coefficient (listed in EPA 1982)
foc = fraction of organic-carbon in the soil
p = bulk density of the soil (gm/cm^3)

The retardation factor represents the mobility of the constituent traveling through the soil in an aqueous solution, relative to the mobility of water. The retardation factor was used in estimating the mass flux of each constituent into the saturated zone.

The mass flux (gm/year) for each constituent was calculated using the following relationship

$$\text{Mass Flux} = \frac{(\text{Leachate Concentration}) \times (\text{Infiltration Rate})}{\text{Retardation Factor}} \qquad (A-4)$$

A.1.2.2 HPS Model

The HPS model is a three-dimensional, transient, analytical solution of the convective-diffusion equation for solute transport in ground water (Galya, 1987). The model incorporates the following features:

- Advection in one dimension with dispersion in three dimensions.
- Simulation of a horizontal plane source in order to correctly model transport from landfills or waste lagoons.
- The ability to perform time-varying as well as steady-state predictions.
- Simulation of the source strength as a mass flux input which can vary with time.
- The effects of retardation and degradation explicitly included in the formulation.

Model Formulation

Transport and first-order degradation of contaminants in an aquifer can be described by the three-dimensional convective-diffusion equation:

$$\frac{\partial C}{\partial t} + \frac{u}{R}\frac{\partial C}{\partial x} = \frac{D_x}{R}\frac{\partial^2 C}{\partial x^2} + \frac{D_y}{R}\frac{\partial^2 C}{\partial y^2} + \frac{D_z}{R}\frac{\partial^2 C}{\partial z^2} - \lambda C + \frac{M_s}{\theta} \qquad \text{(A-4)}$$

where

C = concentration of contaminant
u = ground-water velocity in x direction
t = time
D_x = dispersion coefficient in x direction
$= \alpha_x u$
α_x = dispersivity in x direction
D_y = dispersion coefficient in y direction
$= \alpha_y u$
α_y = dispersivity in y direction
D_z = dispersion coefficient in z direction
$= \alpha_z u$
α_z = dispersivity in z direction
λ = decay or degradation coefficient
R = retardation factor
$= 1 + \rho_b K_d/\theta$
ρ_b = bulk density
K_d = distribution coefficient
θ = porosity
M_s = Mass source flux

Solutions of Equation A-4 for various boundary and source conditions can be specified in terms of the appropriate Green's functions as (Codell and Schreiber, 1977; Yeh and Tsai, 1976; Carslaw and Jaeger, 1959):

$$C = \frac{1}{\theta R} X_o(x,t)\, Y_o(y,t)\, Z_o(z,t)\, T(t) \qquad \text{(A-5)}$$

where

T = degradation function due to degradation or decay of contaminants (includes the effects of biodegradation, hydrolysis, chemical reaction, etc.)

= exp(-λt) for first order degradation (A-6)

X_o, Y_o, Z_o = Green's functions for transport in x,y and z directions, respectively.

Equation A-5 provides the contaminant concentration at any point in space for an instantaneous release of a unit mass of contaminant.

For a horizontal plane source extending from -L/2 to L/2 in the x direction and from -B/2 and B/2 in the y direction:

$$X_o = \frac{1}{2L}\left[\operatorname{erf}\frac{\left(\frac{L}{2} + x - \frac{ut}{R}\right)}{\sqrt{4D_x t/R}} + \operatorname{erf}\frac{\left(\frac{L}{2} - x + \frac{ut}{R}\right)}{\sqrt{4D_x t/R}}\right] \quad (A\text{-}7)$$

where:

L = width of source in x direction
x = distance in X direction to calculation point

$$Y_o = \frac{1}{2B}\left[\operatorname{erf}\frac{\left(\frac{B}{2} + y\right)}{\sqrt{4D_y t/R}} + \operatorname{erf}\frac{\left(\frac{B}{2} - y\right)}{\sqrt{D_y t/R}}\right] \quad (A\text{-}8)$$

where

B = length of source in y direction
y = distance in y direction to calculation point

For a source located at the top of an aquifer of infinite thickness

$$Z_o = \exp(-z^2/(4D_z t/R))/(\pi D_z t/R) \qquad \text{(A-9a)}$$

and for a source located at the top of an aquifer of thickness h

$$Z_o = \frac{1}{H}\left(1+2\sum_{m=1}^{\infty} \exp\left(-\frac{m^2\pi^2 D_z t}{H^2 R}\right)\cos(m\pi z/H)\right) \qquad \text{(6b)}$$

where

z = distance in z direction to calculation point

Substituting Equations A-6 through A-9 into Equation A-5 produces solutions for an instantaneous unit release of mass over an area of length L and width B at the top of an aquifer of infinite or finite thickness. Applying a convolution integral with respect to time provides the concentration C due to a continuous release at a rate M(τ) for any time τ following the beginning of a release.

$$C = \frac{1}{\theta R}\int_0^{\tau} M(\tau)X_o(x,t-\tau)Y_o(y,t-\tau)Z_o(Z,t-\tau)T(t-\tau)d\tau \qquad \text{(A-10)}$$

where

M = mass released per unit time

Equation A-10 is generally applied to predict peak concentrations due to time-varying source terms. Steady-state concentrations can be predicted by specifying a constant source rate M and integrating time from zero to infinity.

Dispersion in the HPS model is formulated as a function of the length scale traveled by the contaminants as follows:

$$\alpha_x = 0.1 X \tag{8}$$

$$\alpha_y = \alpha_x / R_{xy} \tag{9}$$

$$\alpha_z = \alpha_z / R_{xz} \tag{10}$$

where

R_{xy} = Ratio of α_x to α_y
R_{xz} = Ratio of α_x to α_z

and all other parameters are as discussed above.

Equation 8 was suggested by Gelhar and Axness (1981) and Walton (1985) as a means of estimating the longitudinal dispersivity. Mills et al. (1985) suggest that the longitudinal dispersivity should reach some maximum value at large distances from the release point, though they do not propose any specific value. Gelhar et al (1985) present a range of 2.1 - 5.0 for R_{xy} values and a range of 30 - 860 for R_{xz} values.

A.2 Model Applications and Results

A.2.1 Leachate Concentration Modeling

The OLM model was applied to predict the concentration of contaminants leaching out of the lagoon sediment and soil beneath the sediments. The parameters considered are listed in Table A-1. Table A-1 also provides the sediment and soil concentrations of each of these compounds, obtained by averaging measured concentrations taken from the PELA and EPA sampling effort of June 20-24, 1983 (at stations J-05, K-02, and K-03 for sediment and stations L-02A, L-02B, L-05A, L-05B, L-06, L-07, and N-08 for soil); the OLM predictions for sludge and soil; and the solubility for each compound.

TABLE A-1

OLM MODEL PREDICTIONS AND SOLUBILITY VALUES FOR CARCINOGENIC PAH COMPOUNDS AND FLUORANTHENE

(ALL VALUES IN PPM)

Compound	Sludge Conc. in Waste	Sludge OLM Prediction	Soil Conc. in Waste	Soil OLM Prediction	Solubility
Carcinogenic PAH					
Benzo[a]Anthracene/ Benzo[a]Pyrene	510	0.025	15.4	0.0023	0.0089(a)
Benzo[b]Fluoranthene	770	0.013	10.6	0.00073	0.0008
Chrysene	14360	0.137	317	0.010	0.002
Dibenzo[a,h]- Anthracene	560	0.0090	13.8	0.00073	0.0005
Indeno[1,2,3cd]- pyrene	256	0.0038	6.7	0.00032	0.0002
Fluoranthene	9100	0.62	200	0.046	0.26

a. The mean solubility limit of the 2 compounds were used in the model.

As indicated in Table A-1, the OLM model predicts that the sludge leachate concentration for each of the contaminants considered will be greater than the solubility value. Therefore, the OLM model was considered to be invalid and for transport modeling it was assumed that the leachate concentration from the sediment would be equal to the solubility values given in Table A-1.

A.2.2 Model Applications to Live Oak Site

A.2.2.1 Unsaturated Zone

Equation A-2 was used to calculate the moisture content in the unsaturated zone between the lagoon and the upper boundary of the limestone aquifer. In order to apply the equation to the Live Oak Site the following assumptions were made regarding the site surface and ground water conditions:

- The drainage area of the lagoon (A) is 17.3 acres (FTCH 1987)
- THe lagoon area (a) is 3 acres (FTCH 1987)
- The average annual rainfall (I) at the site is 52 inches/year (FTCH 1987)
- The fraction of the total rainfall runoff (C) in the drainage area which infiltrates into the soil, before reaching the lagoon, is 0.45.
- The annual evaporation from the lagoon (E) is 137 acre-in./year (FTCH 1987)
- The steady state flux (q) through the unsaturated zone equals the infiltration rate of lagoon water into the soil and is calculated by

$$q = \frac{C \times I \times A - E}{a}$$

Therefore, q is 89 inches per year for the Live Oak site lagoon.

- The soil of the unsaturated zone can be conservatively assumed to be a 30-foot layer of sand with an organic carbon content of 0.1 percent (foc = .001), where

  Ksat = 1.056 cm/min (Clapp, et al 1978)
  b = 4.05 (Clapp, et al 1978)
  θ_s = 0.4 (Clapp, et al 1978) and
  ρ = 1.5 gm/cm^3

Using the above assumptions, Equation A-3 was used to determine the retardation factor and Equation A-4 was used to determine the mass flux rate for each compound. These values are presented in Table A-2.

A.2.2.2 HPS Model

The HPS model was applied to predict contaminant concentrations at monitoring well MW-8 and at a drinking water supply well located 1600 ft (488 meters) from the lagoon, assumed to be directly downgradient.

Input parameters and assumptions used in the model were:

- the waste area (lagoon) is 70 meters long and 173 meters wide
- ground-water velocity in the x direction is 33.40 meters/year. (Conservative value derived using Darcy's Law; a permeability of 1000 ft/day and a head gradient of 0.0003).
- the dispersivity in the x direction (α_x) is less than or equal to 100 m
- the dispersivity in the y direction is equal to α_x divided by 3 (R_{xy} = 3)

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TABLE A-2

RETARDATION FACTORS AND MASS FLUXES FOR CARCINOGENIC PAH COMPOUNDS, FLUORANTHENE AND PHENANTHRENE

Constituent	Retardation Factor	Mass Flux (gm/yr)
Benzo(a)pyrene/ benzo(a)anthracene	2.1×10^4	0.012
Benzo(b)fluoranthene	4.1×10^3	0.005
Chrysene	1.5×10^3	0.037
Dibenzo(a,h)anthracene	2.5×10^4	0.0005
Indeno(1,2,3-cd)pyrene	1.2×10^4	0.0005
Fluoranthene	2.8×10^2	25.0
Phenanthrene	1.0×10^2	261.0

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- the dispersivity in the z direction is equal to α_x divided by 50 (R_{xz} = 50)
- Mass flux rates were obtained from the unsaturated zone calculations (Table A-2).

The predicted concentrations, after 50 years of constant release from the lagoon, of each constituent at MW-8 and the receptor well are listed in Table A-3, along with the maximum measured concentrations in MW-8. Fluoranthene and phenanthrene were the only constituents detected in MW-8 at concentrations above detection limits. The predicted concentrations of fluoranthene and phenanthrene at MW-8 are 50 and 290 percent greater, respectively, than the measured concentrations at the same well, demonstrating the conservative results of the model.

TABLE A-3

HPS MODEL PREDICTIONS OF CONCENTRATIONS OF CARCINOGENIC PAH, FLUORANTHENE AND PHENANTHRENE AT COMPLIANCE WELL

Constituent	HPS Predicted Conc. at MW-8 (mg/l)	HPS Predicted Conc. at Receptor Well (mg/l)	Highest Measured Conc. at MW-8 (mg/l)
Benzo(a)pyrene/ benzo(a)anthracene	$.1X10^{-5}$	$.6X10^{-7}$	<.020
Benzo(b)fluoranthene	$.6X10^{-6}$	$.2X10^{-7}$	<.020
Chrysene	$.4X10^{-5}$	$.2X10^{-6}$	<.020
Dibenzo(a,h)anthracene	$.6X10^{-7}$	$.2X10^{-8}$	<.020
Indeno(1,2,3-cd)pyrene	$.6X10^{-7}$	$.2X10^{-8}$	<.020
Fluoranthene	.003	.0001	.002
Phenanthrene	.031	.001	.008

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APPENDIX B

DETERMINING SURFACE SOIL CONCENTRATIONS

TABLE B-1

DATA USED FOR DETERMINING SURFACE SOIL CONCENTRATIONS LIVE OAK, FLA.

Source	No.	Carcinogenic PAH	Total Cresote Constituents	PCP	Fluoranthene
EPA, Feb. 1983	TAB. 1-4	17500	221300	3000	29000
LETCO, Sept. 1983 (0-1 ft depth)	A-1	15	32.8	830	3.7
	A-2	0	0	6.1	
	A-3	12.7	43.8	158500	9.6
	A-4	13.9	44.8	985	13.7
	A-5	8.4	23.7		5.1
	A-6	269	1005.3	9550	150
	A-7	29.5	51.3	169.5	7.9
Remedial Investigation Test Holes in Vicinity of Lagoon (0-1 ft, Above Water Only)	5(005)	594	1615	0	490
	8(027)	0	16300	0	7700
	9(034)	3800	37990	0	7300
	10(040)	680	5345	0	2300
	11(046)	166	253.8	0	9.3
	12(051)	11600	146389	0	26000
	13(055)	900	16770	20000	3300
	14(062)	918	2713	0	770
Remedial Investigation Individual Soil Samples in Former Plant Area (0-0.5 ft depth)	300	17.88	32.658	10	0.16
	302	0.919	1.328	0	0.011
	304	15.79	23.724	0	4.7
	306	4.82	8.06	0	0.026
	308	1.18	2.268	0	0.029
	310	0.072	0.0824	0	0.095
	312	1.51	2.934	0	0
	314	19.36	30.736	0	0.35
	316	18.11	33.02	0	3.8
	318	10.7	18.09	0	0

TABLE B-1 (Continued)

Source	No.	Carcinogenic PAH	Total Creosote Constituents	PCP	Fluoranthene
	320	6.77	21.465	0	0
	322	2.02	3.3982	0	0.17
	400	0.503	0.706	0	0
Remedial Investigation Composite Sample of Soil in Wood Storage Area (0-0.5 ft depth)	Area 1	17.7	31.2	0	5.1
	Area 2	11.4	20.09	0	3.1
	Area 3	0.058	0.1515	0	0.036
	Area 4	3.41	6.426	0	1.2
	Area 5	2.91	4.22	0.5	0.58
	Area 6	54.4	77.7	0	5.5
	Area 7	2.5	4.786	0	0.8
	Area 8	1.53	3.38	0	0.73
	MAX	17500	221300	158500	29000
	MEAN	995	12168	5363	2141
	SD	3375	42539	26135	6415
	SEM	555	6993	4356	1069

TABLE B-2

MEAN INDICATOR CHEMICAL CONCENTRATIONS

WHEN TOTAL CREOSOTE SUBSTANCES ABOVE 1000 PPM ARE REMEDIATED

LIVE OAK, FLA.

Source	No.	Carcinogenic PAH	Total Creosote Substances	PCP	Fluoranthene
LETCO, Sept, 1983 (0-1 ft, except as noted)	A-1	15	32.8	830	3.7
	A-2	0	0	6.1	
	A-3	0	0	14.90	0
	A-4	13.9	44.8	985	13.7
	A-5	8.4	23.7		5.1
	A-6	24.1	70.6	805	10.4
	A-7	29.5	51.3	169.5	
RI, Lagoon (0-1 ft, except as noted)	5(007,3-5 ft)	0	0.34	0	0.15
	8(029,3-5 ft)	10.8	105.4	0	23.0
	9(036,3-5 ft)	0	5.09	0	.76
	10(042,3-5 ft)	0.15	13.11	0	.34
	11(046)	166	253.8	0	9.3
	12(054,5-7 ft)	8.63	166.5	0	23
	13(058B,5-7 ft)	6.65	157.2	0	24
RI, Plant Area (0-0.5 ft depth)	300	17.88	32.658	10	0.16
	302	0.919	1.328	0	0.011
	304	15.79	23.724	0	4.7
	306	4.82	8.06	0	0.026
	308	1.18	2.268	0	0.029
	310	0.072	0.0824	0	0.095
	312	1.51	2.934	0	0
	314	19.36	30.736	0	0.35
	316	18.11	33.02	0	3.8

TABLE B-2 (Continued)

Source	No.	Carcinogenic PAH	Total Creosote Substances	PCP	Fluoranthene
	318	10.7	18.09	0	0
	320	6.77	21.465	0	0
	322	2.02	3.3982	0	0.17
	400	0.503	0.706	0	0
RI, Wood Storage Area (0-0.5 ft depth)	Area 1	17.7	31.2	0	5.1
	Area 2	11.4	20.09	0	3.1
	Area 3	0.058	0.1515	0	0.036
	Area 4	3.41	6.426	0	1.2
	Area 5	2.91	4.22	0.5	0.58
	Area 6	54.4	77.7	0	5.5
	Area 7	2.5	4.786	0	0.8
	Area 8	1.53	3.38	0	0.73
	MAX	166	254	985	24
	MEAN	14	36	83	4
	SD	29	56	252	7
	SEM	5	10	44	1

MEMO TO: Distribution
FROM: J. Ryan
DATE: February 17, 1988

RE: ACTION LEVELS FOR THE LIVE OAK SITE

INTRODUCTION

This memorandum discusses action levels for final remediation of the Live Oak site. The document has been prepared based on discussions with EPA and FDER personnel and additional work on the Risk Assessment report for the site. Included in this evaluation are considerations related to: (a) the existing site conditions following the interim action and (b) the proposed biological treatment of the contaminated soils remaining after the interim removal action.

In preparing the action levels the following items were evaluated:

- o criteria for soil concentrations of carcinogenic PAH,
- o background concentrations of carcinogenic PAH,
- o the proposed remedy,
- o leachate concentrations associated with residuals,
- o average site concentrations of carcinogenic PAH,
- o the future development of the site,
- o appropriate risk levels,
- o extent and cost effectiveness of removal, and
- o the risk associated with ingestion of surficial contaminated soils.

SOIL CRITERIA

Although no standards have been set for clean up levels associated with PAH contaminated soil, various guidelines and site specific actions can be used to provide a perspective on soil action levels. The most significant study is by the Center for Disease Control in Atlanta which builds on previous work completed with dioxins.

The Centers for Disease Control's Agency of Toxic Substances Disease Registry (ASTDR) has evaluated the carcinogenic status of polycyclic aromatic hydrocarbons (PAH) relative to 2,3,7,8 tetrachlorodibenzo-p-dioxin, an extremely potent animal carcinogen (3). ASTDR has suggested 100 ppm for PAH in soil as a safe level.

As stated by Dr. Stephen Margolis of the ASTDR:

> "In a published article (4), the Centers for Disease Control (CDC) derived an action level at which to limit human exposure for 2,3,7,8-tetrachloro-dibenzo-p-dioxin (2,3,7,8-TCDD), contaminated residential soil. This derived value was based upon extrapolations from animal toxicity experiments (including carcinogenicity and reproductive effects) to possible human health effects in order to estimate a reasonable level of risk for 2,3,7,8-TCDD. A 10^{-6} excess lifetime risk was used in the development of this TCDD soil level.
>
> The Environmental Protection Agency's Carcinogen Assessment group has derived a relative potency index for more than 50 chemicals (5). The order of magnitude potency index for 2,3,7,8-TCDD is eight, while that for benzo(a)pyrene is only three. Thus, 2,3,7,8-TCDD is considered to be five orders of magnitude more potent as a carcinogen than benzo(a)pyrene. Using only this order of magnitude difference in potency between the two chemicals and the CDC-derived residential soil action level, gives 100,000 ppb of benzo(a)pyrene equivalent to 1 ppb of 2,3,7,8-TCDD in soil."

This comparison used benzo(a)pyrene as the representative PAH. It must be recalled that benzo(a)pyrene is the most potent of all PAH compounds studied. Therefore, this model is considered to be a conservative model when applied to the other suspected carcinogens.

An additional measure of conservatism is added to the ASTDR model by overestimating the ingestion of contaminated soil. Again, as stated by Dr. Margolis,

> "In the model used to derive the 2,3,7,8-TCDD soil value, the assumption concerning the amount of soil ingested has been shown to be high. A recent published study by CDC has shown the amount of soil ingested by children of the soil-eating age ranges from 0.1 to 1 gram per day (S. Binder personnel communication). Thus, the model estimate for soil

> ingestion during the period of minimum hygiene is excessive by at least an order of magnitude. Since the other soil ingestion rates in the model are also estimates, there is a good likelihood that they are also in error, possibly by more than an order of magnitude. Thus, the model very likely overestimates the total lifetime soil ingestion exposure by at least one order of magnitude."

Because of the conservative nature of the recommended CDC action levels, USEPA Region VI and Region VII have used the 100 ppm as action levels for carcinogens at PAH contaminated sites.

BACKGROUND CONCENTRATIONS

The remedial investigation included sampling of background areas where PAH compounds may have been used or occur naturally. One area was a swamp and the other was abandoned railroad tracks. Concentrations of carcinogenic PAH at the abandoned railroad tracks were in excess of 16 ppm and in the swamp were 0.8 ppm.

PROPOSED REMEDY

The proposed remedy is described in the attached memorandum entitled "Conceptual Plan for Final Site Remediation". The remedy involves: (a) plant demolition, (b) removal and off site disposal of hardened creosote sludges from the plant area, (c) excavation of contaminated soils from the plant area, (d) biological treatment of contaminated soils within or adjacent to the former lagoon and (e) site security and monitoring.

It should be noted that the majority of site contamination has been removed as part of the interim removal action. Over 15,000 tons of sludges and contaminated soils were removed from the lagoon. (See memo on Interim Removal Action.) An additional 1500 tons of hardened creosote will be removed from the plant site and disposed at CWM's facility in Emelle, Alabama, as part of the proposed remedy. An estimated 10,000 tons of soil containing greater than 1000 ppm total creosote substances will be treated on-site biologically. The treatment goal for these soils is 100 ppm carcinogenic PAH. Based on modeling and experience with biological treatment of similar materials it is estimated that the treatment process will take less than two years.

The cost effectiveness of the proposed plan can be contrasted to the cost effectiveness of removing soils containing less than 100 ppm carcinogenic PAH by comparing the overall mass of TCS removed (or treated). Approximately 6,000 tons of sludge and 9000 tons of highly contaminated soil were removed as part of the interim removal. It is estimated that another 1500 tons of hardened creosote will be removed from the plant area and disposed of at Emelle and 10,000 tons of moderately contaminated soil will be treated as part of the final remedial action. The sludge and hardened creosote are estimated to have an average concentration in excess of 100,000 ppm total creosote substances (TCS). The highly contaminated soil is estimated to be in excess of 10,000 ppm TCS and the moderately contaminated soil is estimated to have TCS concentrations ranging from 1000 to 5,000 ppm TCS.

Surficial soil which is greater than 100 ppm TCS and less than 1000 ppm TCS was not identified in the R.I. Surficial soil which exceeds 10 ppm TCS and is less than 100 ppm TCS is estimated at approximately 7500 tons. Surficial soil which exceeds one ppm TCS but is less than 1,000 ppm TCS is estimated at 31,750 tons.

Table 1 compares the mass of TCS which will have been removed and the mass of TCS which will be treated to the mass present in the surficial soil which is in excess of one ppm in the surficial soils. The cost of removing or treating these materials is expressed on the basis of $/pound of TCS. It can be seen that the incremental cost of removing the surficial soils is two orders of magnitude greater despite the fact that these materials represent less than 0.03 percent of the total creosote substances.

LEACHATE MIGRATION

This pathway was evaluated in detail in the existing Risk Assessment report and was concluded to represent an acceptable risk under the existing conditions in the lagoon. This pathway is revisited, however, to evaluate the impact of the treatment drainage returning to the pond. The RITZE model was used to evaluate the concentrations of carcinogenic PAH in the soil pore water which would be recycled through the treatment pond. The modeling results indicate that this water should be below detection limits of one ppb for individual PAH compounds.

These results are reasonable in light of reported sediment/water partition coefficients for these compounds. Tables 2A and 2B present the range of these partition coefficients. In general, log Koc range from 5.29 to 7.34. The actual water concentration will be a function of the soil organic carbon content and the concentration of the individual compound. Using an organic carbon content of 0.5 to one percent and an initial concentration of 20 ppm, the estimated water concentration would range from 21 to <0.5 ppb.

These results have also been shown in TCLP testing of the contaminated soil and sludges. These results are shown on Table 3 and are expressed as the total concentration and the TCLP (water soluble) extract.

Table 1
COST EFFECTIVENESS COMPARISON

MATERIAL	TONS MATERIAL	TCS (ppm)	TONS TCS	$/TON MATERIAL	$/POUND TCS	% TCS REMOVED	TOTAL COST ($K)	% TOTAL COST
SLUDGE	6000	100000	600	200	1	67.39	1200	21.1
HIGHLY CONTAMINATED SOIL	9000	10000	90	200	10	10.11	1800	31.65
HARDENED CREOSOTE	1500	100000	150	200	1	16.85	300	5.27
MODERATELY CONTAMINATED SOIL	10000	5000	50	50	5	5.62	500	8.79
SOIL >1 PPM TCS	37750	9	0.34	50	2775	0.03	1887.5	33.19
TOTAL	64250		890.34			100	5687.5	100

TABLE 2a
RANGE OF VALUES FOR LOG(KOC) FOR SELECTED PAH COMPOUNDS (a)

COMPOUND	REFERENCE NUMBER 6	7(b) lower	 upper	8	(c) lower	 upper	RANGE lower	 upper
BENZO(A)ANTHRACENE		6.27			5.29	5.29	5.29	6.27
CHRYSENE	5.40				5.29	5.47	5.29	5.47
BENZO(B)FLUORANTHENE	5.36				6.25	6.25	5.36	6.25
BENZO(K)FLUORANTHENE	5.63				6.52	6.52	5.63	6.52
BENZO(A)PYRENE	5.83	6.65			5.72	5.72	5.72	6.65
DIBENZO(A,H)ANTHRACENE	5.76	5.75	6.49	6.22	5.65	5.65	5.65	6.49
INDENO(1,2,3-CD)PYRENE	6.45				7.34	7.34	6.45	7.34

(a) All units for partition coefficients are ml/g.

(b) Data was derived from Kd data -
Kd = Koc * (organic carbon fraction)

(c) Calculated from Kow data - (9,6)
and equation: (10, 11)
log Koc = log Kow - 0.317

TABLE 2b
RANGE OF LEACHATE WATER CONCENTRATIONS

COMPOUND	Koc (1)		WATER CONCENTRATION (ug/l) (2) 0.5% organic carbon		1.0% organic carbon	
	lower	upper	upper	lower	upper	lower
BENZO(A)ANTHRACENE	194984	1862087	21	2	10	1
CHRYSENE	194984	295121	21	14	10	7
BENZO(B)FLUORANTHENE	229087	1778279	17	2	9	1
BENZO(K)FLUORANTHENE	426580	3311311	9	1	5	1
BENZO(A)PYRENE	524807	4466836	8	1	4	0
DIBENZO(A,H)ANTHRACENE	446684	3090295	9	1	4	1
INDENO(1,2,3-CD)PYRENE	2818383	21877616	1	0	1	0

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(1) From Table 2a
(2) calculated using a constituent concentration of 20 ppm.

TABLE 3

Concentrations of Indicator Parameters
Analyzed in Soil Samples
taken August 1986 from test pits
within the Lagoon during low water level
(concentration in ppm)

Sample Number	02	03	04	02	03	04
Test Pit Number	1	2	3	1	2	3
Soil Type	Sand	Sand	Sludge & Sand	Sand	Sand	Sludge & Sand
Visible Contaminated?	No	Yes	Yes	No	Yes	Yes
Depth (feet)	1-2	1-3	0-0.5	1-2	1-3	0-0.5
PARAMETER:	---- Total Concentrations ----			----- TCLP Concentrations ----		
Naphthalene	<0.15	4	23000	<0.01	0.09	11
Acenaphthylene	<0.15	<0.15	230	<0.01	<0.01	<0.01
Acenaphthene	0.33	23	8600	0.01	0.03	0.67
Fluorene	0.33	29	11000	0.01	0.24	0.44
Pentachlorophenol	<0.15	<0.15	990	<0.01	<0.01	<0.01
2,4,6-Trichlorophenol	<0.15	<0.15	<100	<0.01	<0.01	<0.01
Phenanthrene	0.41	75	24000	<0.01	0.27	0.47
Anthracene	<0.15	9.70	13000	<0.01	0.02	0.08
Fluoranthene	0.63	36	9400	<0.01	0.03	0.42
Pyrene	0.45	24	7400	<0.01	0.02	0.09
Benzo(a)anthracene	<0.15	5.1	1800	<0.01	<0.01	<0.01
Chrysene	<0.15	4.9	1100	<0.01	<0.01	<0.01
Benzo(b)fluoranthene	<0.15	3.7	600	<0.01	<0.01	<0.01
Benzo(k)fluoranthene	<0.15	1.2	400	<0.01	<0.01	<0.01
Benzo(a)pyrene	<0.15	1.5	370	<0.01	<0.01	<0.01
Indeno(1,2,3-cd)pyrene	<0.15	0.43	110	<0.01	<0.01	<0.01
Dibenzo(a,h)anthracene	<0.15	0.27	<100	<0.01	<0.01	<0.01
Benzo(g,h,i)perylene	:0.15	0.38	100	<0.01	<0.01	<0.01
Total Creosote Substance	2.15	218.18	101110	0.02	0.70	13.17
Total Pentachlorophenol	<0.15	<0.15	990	<0.01	<0.01	<0.01
Total Non-Carcinogenic PAH	2.15	202.28	97130.00	0.02	0.70	13.17
Total Carcinogenic PAH **	<0.15	15.90	3980.00	<0.01	<0.01	<0.01
Percent Carcinogens of Total Creosote	0%	7%	4%	0%	0%	0%

Notes: Analysis by Rocky Mountain Analytical Laboratory.
Refer to RMAL # 61897
N.D. indicates not detected
Carcinogenic PAHs: Benzo(a)anthrac[illegible]. Benzo(b)fluoranthene, Chrysene
Benzo(a)pyrene, Dibenzo(a,h)anthra[illegible]e, Indeno(1,2,3-cd)pyrene

AVERAGE SITE CONCENTRATIONS

The exposure assessments related to soil ingestion consider a long term site exposure rather than a short term exposure related to a specific area on the site. Any development of the site for either industrial or residential purposes would require extensive earthwork. It is appropriate, therefore, to consider a composite site concentration of carcinogenic PAH. This composite is based on an average concentration across the site to a depth of two feet.

The average concentrations have been calculated based on the conditions which will exist after the contaminated soil from the plant area have been removed and assumes the residuals in the former lagoon have been treated to a level less than 100 ppm carcinogenic PAH.

Figure 1 shows the areas used in these calculations. The former wood storage area is approximately 520,000 square feet in size. The plant area is approximately 240,000 square feet in size and the former lagoon area (and future treatment area) is approximately 240,000 square feet in size. The overall site area is about 2,100,000 square feet in size.

Average concentrations over the plant area and the wood storage area were calculated using existing surficial data (0-12 inches) excluding highly contaminated samples from the plant area which will be removed as part of the final remedial action. Concentrations from 12 to 24 inches were estimated using 50 percent of the concentration from six to 12 inches. If the concentration measured at six to 12 inches was less than 1.3 ppm then the concentration at 12 to 24 inches was assumed to be BDL. Figure 1 shows the location of the sampling points and Table 4 presents the measured concentrations and the estimated composite average.

Concentrations in the proposed treatment area were assumed to be 100 ppm in the upper 12 inches and 10 ppm at 12 to 24 inches based on modeling results. (See the memorandum "Conceptual Plan for Final Site Remediation". The composite concentrations for the three areas is 16 ppm and for the overall site is 7.6 ppm. for carcinogenic PAH.

SITE DEVELOPMENT

Most of the soil criteria revolve around the issue of the future use of the site. The PRP's are pursuing institutional controls which would preclude the use of the site as a residential development. The action levels, therefore consider both unrestricted residential development and restricted site access

TABLE 4

AVERAGE CONCENTRATIONS OF CARCINOGENIC PAH (ppm)

WOOD STORAGE AREA

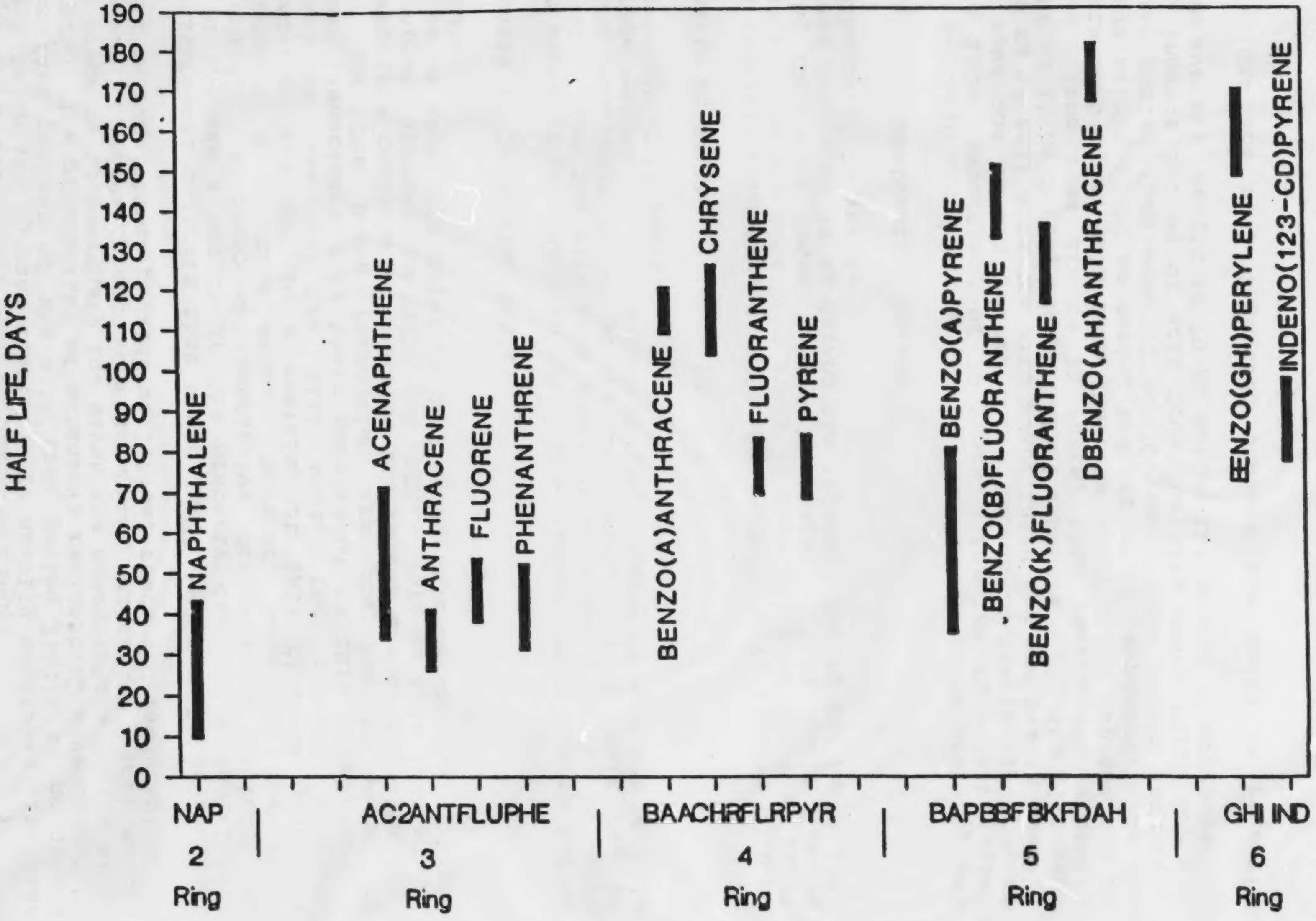
SAMPLE NUMBER	0-6 INCHES	6-12 INCHES	12-24 INCHES
1.00	11.40	0.23	BDL
2.00	11.40	0.23	BDL
3.00	11.40	0.23	BDL
4.00	11.40	0.23	BDL
5.00	2.90	0.06	BDL
6.00	2.90	0.06	BDL
7.00	2.90	0.06	BDL
8.00	2.90	0.06	BDL
9.00	2.80	0.70	BDL
10.00	2.80	0.70	BDL
11.00	2.80	0.70	BDL
12.00	2.80	0.70	BDL
13.00	3.40	1.30	BDL
14.00	3.40	1.30	BDL
15.00	3.40	1.30	BDL
16.00	3.40	1.30	BDL
17.00	1.50	0.15	BDL
18.00	1.50	0.15	BDL
19.00	1.50	0.15	BDL
20.00	1.50	0.15	BDL
21.00	62.00	6.40	3.20
22.00	62.00	6.40	3.20
23.00	62.00	6.40	3.20
24.00	62.00	6.40	3.20
25.00	17.70	7.10	3.60
26.00	17.70	7.10	3.60
27.00	17.70	7.10	3.60
28.00	17.70	7.10	3.60
29.00	0.06	BDL	BDL
30.00	0.06	BDL	BDL
31.00	0.06	BDL	BDL
32.00	0.06	BDL	BDL
avg	12.72	1.99	0.85
composite avg. (over 2 feet)		4.10	

PLANT AREA

SAMPLE NUMBER	0-6 INCHES	6-12 INCHES	12-24 INCHES
33.00	17.20	0.73	BDL
34.00	0.92	BDL	BDL
35.00	15.80	6.60	3.30
36.00	4.80	0.62	BDL
37.00	1.20	0.04	BDL
38.00	0.07	0.05	BDL
39.00	1.50	0.02	BDL
40.00	19.40	0.30	BDL
41.00	18.10	11.40	5.70
42.00	10.70	0.06	BDL
43.00	6.70	0.02	BDL
44.00	1.60	2.10	1.00
45.00	0.50	BDL	BDL
avg.	7.58	1.69	0.77
composite avg. (over 2 feet)		2.70	

FIGURE 1

PAH HALF LIVES - LITERATURE REVIEW DATA
Min - Max Ranges



(such as an industrial park or preserve land). The site in its current condition is heavily vegetated with less than five percent of the site area having little or no ground cover. The probability of extensive residential development on the property is extremely low given the demographics of the area. However, if development were to occur, extensive earth moving would be required to provide utilities, roads and landscaping.

Institutional controls that would limit residential development could include a notice in the property deed or a restrictive covenant. A proposed restrictive covenant is included as Attachment A to this memo. However, Amax and The Brown Foundation are not in a position at this time to guarantee restricted use of the site until the current property owners/lienholders give their permission to this arrangement. Amax and The Brown Foundation are prepared to make the appropriate contacts to restrict the future use of the site upon conceptual approval by USEPA and FDER. Various site development scenarios were considered in developing this memorandum. These include:

Scenario 1: Industrial Adult

This scenario considers an adult who inadvertently ingests 5.0 mg of soil from the site once every 30 days all year for 35 years during adulthood (ages 25 to 60 years). This adult has an average weight of 70 kg during the exposure and has a life expectancy of 70 years.

Scenario 2: Neighborhood Child

This scenario considers a child who inadvertently ingests 50.0 mg of soil from the site once every 30 days all year for five years during childhood (ages 6 to 11 years). This child has an average weight of 30 kg during the exposure period and has a life expectancy of 70 years.

Scenario 3: Residential Person

This scenario considers a person who lives on the property and has a life expectancy of 70 years. For the five-year period from ages one to six years, this person inadvertently ingests 100.0 mg of soil from the site every third day and has an average weight of 15 kg during the period of exposure. For the five-year period from ages six to 11 years, this person inadvertently ingests 50.0 mg of soil from the site every third day and has an average weight of 30 kg during the period of exposure. For the 59-year period from ages 11 to 70 years, this person inadvertently ingests 5.0 mg of soil from the site every third day and has an average weight of 70 kg during the period of exposure.

The weight and soil ingestion factors are based on recently published data (12,13). No specific criteria are available in terms of the frequency of exposure. Due to the relative inaccessibility of the site and the improved hygiene of adults, the frequency of exposure for the neighborhood and industrial setting is reasonable. This frequency is increased by an order of magnitude (10 times a month) for the residential setting. This also is reasonable in light of the conservative lifetime (0-70 years) which the assessment spans, and considerations related to:

- Inclement weather
- close supervision of young children, and
- time spent away from the home (at school, work, etc.).

Summary: Limited exposure currently exists due to contact with exposed surface soil. The probability of residential development is extremely low in the foreseeable future. Institutional controls restricting access are feasible.

RISK LEVELS

A major consideration in establishing action levels is the appropriate level of risk. CERCLA guidance recommends risk levels which range between 10^{-4} to 10^{-7}. Generally, a level of 10^{-5} to 10^{-6} is considered appropriate for protection of human health. The lower risk (10-6) is appropriate in instances where the site conditions represent a substantial and immediate threat to human health and the environment. The 10^{-5} risk is appropriate for situations such as Live Oak where the probability of risk is low and is driven by conservative modeling assumptions and future land use considerations.

SOIL INGESTION

The Risk Assessment report provided an estimate of risk for the site based on a number of highly conservative assumptions and concluded that the baseline risk associated with the current conditions (assuming waste removal to 1000 ppm of total creosote constituents) was acceptable. This assessment is revisited to evaluate risk the associated with both industrial and residential development. Important assumptions made in this assessment include:

THE POTENCY OF FACTOR FOR BENZO(A)PYRENE: The "Superfund Public Health Evaluation Manual", (1) conservatively estimates a cancer potency factor (CPF) for benzo(a)pyrene of 11.5 $[\text{mg/kg/day}]^{-1}$. ICF Clements (the same contractor which prepared the above Guidance Manual) has completed a recent evaluation of existing data and the application of a biologically based dose response to evaluate the potency of benzo(a)pyrene; (2) This evaluation has established the potency of benzo(a)pyrene as 5.74 $[\text{mg/kg/day}]^{-1}$. This assessment is provided as Attachment B to this memorandum.

THE RELATIVE POTENCY OF THE SUSPECTED CARCINOGENS: The existing site database was reviewed to evaluate the relative ratios of the carcinogenic PAH. The data base included the soil data on the plant area and former wood storage area summarized in Table 4. The data base indicates the average relative proportions of the carcinogenic PAH are:

Compound	Relative % of Total Carcinogens	CPF $(\text{mg/kg/day})^{-1}$
benzo(a)pyrene	11.54%	5.74
benzo(a)anthracene	8.71%	0.8323
benzo(b)fluoranthene	49.43%	0.8036
chrysene	18.52%	0.0253
dibenzo(a,h)anthracene	3.56%	6.3714
indeno(1,2,3-cd)pyrene	8.23%	1.3317

Shown above is the cancer potency factor calculated by ICF Clemens for the other carcinogens. The relative distribution of the carcinogens and their cancer potency factor was used in modeling risks associated with soil ingestion. The cumulative cancer potency factor based on the relative distribution of the carcinogens is 1.4713.

MATRIX/AVAILABILITY EFFECTS: The bioaccumulation of the PAH compounds is strongly related to the soil matrix containing the compounds. Low levels of compounds sorbed onto soil cannot be readily accumulated by the body. A matrix factor of 0.3 is used in this assessment and is consistent with the value used by the CDC in evaluating action levels for dioxin contaminated soil.

DEGRADATION FACTORS: A half-life of 0.5 years was used to model the photochemical and biological degradation of the carcinogens. Attachment C presents a database which shows these carcinogens generally have half lives less than 0.5 years. Figure 1 presents the maximum range associated with the 95 percent confidence interval for the attached data base. The half year assumption is the maximum value for any of the carcinogenic compounds evaluated.

RELATIVE RISK

The relative risk associated with the residuals remaining on the plant site after completion of the proposed remedy was calculated for each of the relevant areas as well as for the site as a whole. Table 5 presents the average concentration associated

TABLE 5

LIFETIME RISK ASSOCIATED WITH VARIOUS AREAS OF THE SITE AFTER TREATMENT OF CONTAMINATED SOILS TO 100 ppm TOTAL PAH CARCINOGENS

	Risk Levels Under Various Scenarios		
ocation	Residential (b)	Industrial	Neighborhood Child
ood Storage Area	7.7×10^{-8}	8.2×10^{-11}	1.9×10^{-9}
lant Area	5.0×10^{-8}	5.4×10^{-11}	1.3×10^{-9}
reatment Area	1.0×10^{-6}	1.1×10^{-9}	2.6×10^{-8}
ite Area (a)	1.4×10^{-7}	1.5×10^{-10}	3.6×10^{-9}

) weighted average of wood storage area, plant area, treatment area and the overall site.

) Note: The residential scenario assumes that houses are constructed on the site immediately after treatment is completed and that a person spends his entire life on this site (from birth to 70 years). This scenario is extremely conservative and highly unlikely given the demographics of the area.

with each area (weight averaged over two feet) and the associated risk level with various exposure scenarios. The table shows that the risks associated with the proposed remedy range from 10^{-7} to 10^{-10} for the overall site. This risk is based on the completion of the proposed remedy which includes treating the contaminated soils down to a level of 100 ppm total carcinogens. Attachment D provides the equations used in these calculations.

SUMMARY

This review has evaluated a variety of considerations which affect soil action levels for the Live Oak site. The action levels have considered both average site concentrations as well as maximum concentrations for individual areas.

The proposed maximum concentrations for surfical soils in any given area is 100 ppm of carcinogenic PAH. This value is based on work completed by the Center for Disease Control on the relative potency of PAH carcinogens as compared to 2,3,7,8-TCDD. This value has been used as an action level at other PAH contaminated sites.

Under the proposed action levels, the maximum concentration criteria would be applied to the treatment area. The remainder of surficial soils on the site are well below the proposed maximum criteria. The risk levels on a site average basis following treatment to 100 ppm would be 10^{-7} for an unrestricted development scenario and 10^{-9} for a restricted development scenario. Therefore, no further action is necessary for site soils below 100 ppm carcinogenic PAH. It should be noted that the unrestricted development scenario assumes that houses are constructed on the site immediately after the completion of the proposed remedy and that individuals live on the site from birth to age 70. Given the demographics of the area, this scenario is highly unlikely. Even so, the associated risk levels are 10^{-7} which demonstrates the proposed remedy is highly protective of human health and the environment.

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COMPARATIVE POTENCY APPROACH FOR ESTIMATION OF THE TOTAL CANCER RISK ASSOCIATED WITH EXPOSURES TO MIXTURES OF POLYCYCLIC AROMATIC HYDROCARBONS IN THE ENVIRONMENT

FINAL REPORT

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July 1, 1987

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APPENDIX A

APPENDIX B

These estimates represent reductions of 50% for ingestion exposure and 90% for inhalation exposure as compared to EPA's estimates. The relative potency estimates for other carcinogenic PAHs were found on the basis of data from nine experimental studies to range from 0.0044 to 4.50 as compared to B[a]P. The estimates based on the most reliable of the studies were selected and are summarized in Table IV-26; this table is reproduced below. The estimates were found not to vary to a great extent among studies for a particular PAH.

The results of this study indicate that to estimate the cancer risk due to a mixture of PAHs in the environment, the concentrations of carcinogenic PAHs should be measured and used in conjunction with relative potency values to estimate the total risk.

SUMMARY OF RELATIVE POTENCY ESTIMATES DERIVED FOR PAHs

Benzo[a]pyrene	1.0
Benz[a]anthracene	0.145[b]
Benzo[b]fluoranthene	0.140[a]
Benzo[k]fluoranthene	0.066[a]
Benzo[ghi]perylene	0.022[a]
Chrysene	0.0044[c]
Dibenz[ah]anthracene	1.11[c]
Indeno[1,2,3-cd]pyrene	0.232[a]

[a] Deutsch-Wenzel et al. (1983).

[b] Bingham and Falk (1969).

[c] Wynder and Hoffmann (1959).

TECHNICAL SUMMARY AND CONCLUSIONS

A relative potency approach for estimation of the carcinogenic risk of complex mixtures of polycyclic aromatic hydrocarbons (PAHs) was developed as an alternative to the current practice of assuming that all carcinogenic PAHs are equivalent in potency to benzo[a]pyrene, which has little scientific support. B[a]P has consistently been demonstrated to be one of the most potent carcinogenic PAHs to which humans might be expected to be exposed environmentally. As a result, estimates of cancer risk using a B[a]P one-to-one equivalency approach will greatly overestimate the carcinogenic potency of most mixtures of PAHs. Use of a relative potency approach that takes into account the differing potencies of carcinogenic PAHs would yield a more realistic estimate of risk, with a sounder biological basis.

In this report, a new method is developed for estimating the cancer risk associated with exposure to mixtures of PAHs that attempts to rectify the problems inherent in earlier approaches in use by EPA and others. This study focuses on three critical elements. First, a biological paradigm was proposed for PAH-induced carcinogenesis. Such a paradigm is essential to provide a biological rationale for the dose-response model and to account for any interactive effects (i.e., synergistic or antagonistic) that might be postulated. Second, a two-stage mathematical model was postulated that is consistent with the paradigm. The model parameters were estimated using rodent tumor response data following exposure to B[a]P. The two-stage model is a special case of the Moolgavkar and Knudson (1981, 1986) cancer risk model that was adopted by Thorslund et al. (1987) to account for exposure to known levels of carcinogenic agents. The model may also be viewed as a special case of the classic Armitage and Doll (1954) multistage model in its time-independent form, and a restricted case of the Armitage and Doll (1957) two-stage model in both its time-independent or -dependent forms. The advantages of the model are that it is based on a strong theoretical argument derived from biological principles yet is simple enough to obtain estimates for its parameters using very limited data. Using this model, the estimate of cancer potency for B[a]P was lowered by 50% for ingestion exposure and by 90% for inhalation exposure as compared to EPA's estimates.

The third critical element in the development of the method was obtaining estimates of relative potency for carcinogenic PAHs other than B[a]P using the structural form of the previously derived model. This aspect depended upon using bioassay results that were obtained from systems that are not suitable for direct extrapolation to humans because the routes of exposure employed are not comparable to those by which humans are exposed in the environment. When carcinogenic PAHs and B[a]P have been tested concurrently, however, such experiments can be used to estimate the relative potencies of other PAHs compared to B[a]P. These relative potencies have a specific biological interpretation: they provide an estimate of the ratio of exposure-induced mutation rates per unit of

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exposure. Under the assumptions of the model, these mutations are thought to transform a normal stem cell into a preneoplastic cell and a preneoplastic cell into a malignant cell. Using nine experimental studies, estimates of potency for carcinogenic PAHs were obtained that ranged from 0.0044 to 4.50 as compared to B[a]P. The estimates were consistent among studies for a particular PAH. Those that were obtained from the most reliable studies are summarized in Table IV-26 (and on page vi).

Once the relative potency estimates were obtained, they were used in conjunction with the B[a]P dose response model that was derived and the dose additivity assumption to obtain estimates of cancer risk associated with any specified exposure to multiple PAHs. The dose additivity assumption is synonymous with what is referred to by Finney (1964) as simple similar action and advocated for use in the EPA (1986b) guidelines for assessing the cancer risk of mixtures when inadequate data are available. Simple similar action implies that all carcinogenic agents in a mixture induce carcinogenesis by similar mechanisms. Since carcinogenic PAHs appear to be metabolized to similar reactive derivatives, produce comparable adducts with DNA and the same types of tumors in experimental animals, the assumption of a common mechanism of action (i.e., dose additivity) is plausible.

Under the assumption of dose additivity, the cancer risk due to exposure to multiple PAHs can be obtained as follows. The total exposure units equivalent to B[a]P in a mixture to which an individual is exposed is calculated by taking the sum of the products of the relative potencies and the exposure levels for each PAH. These B[a]P-equivalent exposure units are then substituted into the dose response model for B[a]P to obtain the cancer risk associated with exposure to the PAH mixture. This procedure is evaluated in this document using bioassay results from an experiment conducted by Schmahl et al. (1977) using two different mixtures of PAHs. The resulting predictions were encouragingly close to the tumor rates observed.

For the sake of comparison, more familiar but less defensible models from a biological theory veiwpoint have also been used in this report to estimate relative potencies. Results reasonably comparable to those from the two-stage model were obtained using the one-hit and linearized multistage models, suggesting that the results are not highly model-dependent.

This study has demonstrated that estimation of the total cancer risk due to exposure to a mixture of PAHs should be based on measurements of the concentrations of its carcinogenic components and relative estimates of their potency.

I. INTRODUCTION

The majority of occupational and environmental chemical exposures are to complex mixtures. Emissions from combustion sources and leachates from hazardous waste sites are examples of complex mixtures that contain hundreds of components that have not been completely characterized. Estimating the potential human health hazards associated with such mixtures is difficult because of inadequate chemical and toxicological characterization, as well as the extent of heterogeneity between similar mixtures from different sources.

For complex mixtures of polycyclic aromatic hydrocarbons (PAHs) such as those found at hazardous waste sites, the standard approach to estimating the human cancer risk associated with exposure has been to assume that the carcinogenic PAH components (excluding heterocyclic compounds such as the nitroaromatics) are equivalent by weight to benzo[a]pyrene (B[a]P) in terms of their carcinogenic potency (EPA 1980, 1984). Based on a limited chemical characterization of the mixture, a number of PAHs are identified and their contributions to cancer risk are estimated based on their relative concentrations in the mixture, their expected contributions to human exposure, and the statistical upper-bound on the potency of B[a]P. This procedure has several serious problems.

The assumption that all the carcinogenic PAHs have potencies equivalent to that of B[a]P is not supported by the scientific literature. In studies that have been performed to

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evaluate the comparative potencies of PAHs, B[a]P has consistently proved to be one of the most potent carcinogens tested. These studies will be described in detail in Section IV. Studies of the carcinogenic potencies of complex mixtures of PAHs as compared with B[a]P have shown that most mixtures are considerably less potent than B[a]P alone. For example, various kinds of combustion emissions and B[a]P were tested for potency as tumor initiators on the skin of SENCAR mice (Slaga et al. 1980). Table I-1 shows that the PAH mixtures were much less potent as tumor initiators than B[a]P. The authors calculated relative potency estimates that ranged from 0.007 for coke oven emissions extract to less than 0.002 for diesel engine exhaust extract, using papillomas/mouse-mg as the end point. In another study, the tumorigenicity of an automobile emission condensate (AEC), a diesel emission condensate (DEC), a representative mixture of carcinogenic PAHs, and B[a]P were tested for carcinogenicity by chronic application to mouse skin (Misfeld 1980). The results are shown in Table I-2. Relative potencies were calculated by the authors to be 0.00011, 0.0053, and 0.36 for DEC, AEC, and the PAH mixture, respectively, as compared to B[a]P. Such results make the use of the assumption that all carcinogenic PAHs are as potent as B[a]P to predict the total carcinogenicity of PAH mixtures difficult to support.

Another problem with the EPA's use of B[a]P as a surrogate for the components of PAH mixtures is that it assumes that the total cancer risk of the mixture can be predicted on the basis

TABLE I-1

RELATIVE TUMOR-INITIATING POTENCY OF VARIOUS EMISSION EXTRACTS AND BENZO[a]PYRENE

Substance[a]	Relative Potency[b] Based on (papillomas/mouse-mg)
Benzo[a]pyrene	1.0
Roofing-tar emission extract	0.004
Coke-oven emission extract	0.007
Caterpillar diesel exhaust extract	[c]0
Oldsmobile diesel exhaust extract	0.002
Nissan diesel exhaust extract	0.007
Mustang gasoline-engine exhaust extract	0.002
Cigarette-smoke condensate	[c]0

[a] Material was applied to Sencar mice once as initiator. Phorbol myristate acetate (2 ug), twice a week, was used as promoter. Emission exposures that were used in the relative potency calculations were restricted to the linear portion of the dose-response curve.

[b] As calculated by authors.

[c] These entries refer to potencies that were not significantly different from zero at $p=0.05$.

Sources: Slaga et al. (1980) and NAS (1983).

TABLE I-2

CARCINOGENIC ACTIVITY OF AUTOMOBILE EMISSION CONDENSATE (AEC), DIESEL EMISSION CONDENSATE (DEC), AND PAHs ON MOUSE SKIN

Treatment	%Mice with Tumors	Relative Potency[b]
Solvent control	0	
Benzo[a]pyrene:		1
3.86 ug	32.8	
7.69 ug	60.9	
15.4 ug	89.1	
AEC:[a]		0.0053
290 ug	10.3	
880 ug	44.3	
2,630 ug	83.3	
DEC:[a]		0.00011
4,300 ug	0	
8,600 ug	2.6	
17,150 ug	12.7	
Mixture of PAHs:		0.36
3.5 ug	1.3	
10.5 ug	38.7	

[a] Obtained with leaded fuel.

[b] As calculated by authors.

Sources: Misfeld (1980) and NAS (1983).

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of response additivity. The EPA guidelines for assessing the cancer risk of chemical mixtures in the absence of combined exposure data, however, advocate the assumption of dose additivity (1986b). Dose additivity is synonymous with what is referred to by Finney (1964) as simple similar action. When agents induce cancer by the same mechanism, they are characterized by simple similar action and the cancer risk due to combined exposure can be obtained by estimating the risk due to the total additive dose of the agents.

To use this method for mixtures of PAHs, estimates of the relative potencies of the components are required. The relative potency of the jth carcinogenic PAH compared to the potency of B[a]P at response level p can be defined as

$$R_j(p) = x(p)/y_j(p)$$

where $x(p)$ and $y_j(p)$ are the number of exposure units of B[a]P and the jth carcinogenic PAH, respectively, required to produce a total carcinogenic response rate of p in the test system.

If the mechanism of action of B[a]P is the same as that of the jth carcinogenic PAH, it follows that $R_j(p) = R_j$ (i.e., the relative potency is independent of the response level).

Under the hypothesis of simple similar action, it follows directly that the joint response to a set of carcinogenic PAHs is dependent upon the total PAH exposure, T, expressed in units equivalent to B[a]P. This exposure may be written as

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$$T = \sum_{j=1}^{m} R_j y_j + x \qquad \text{(I-1)}$$

ere

m = the total number of indicator PAHs exclusive of B[a]P;

y_j = the exposure to the jth indicator PAH;

x = exposure to B[a]P; and

R_j = relative potency of the jth indicator PAH compared to B[a]P.

The probability of a cancer response given T is P(T), where P(˙) is the dose-response relationship for B[a]P. For this approach to be valid, the function P(˙) must be monotonic in the experimental range with values of x and y_j expressed in the same units.

In the case of mixtures of carcinogenic PAHs, dose additivity has been postulated to be a reasonable assumption because they appear to be metabolized to similar reactive derivatives that interact with DNA and produce similar tumors (IARC 1983). Kaden et al. (1979) found a simple additive relationship of the mutagenicity of soot and added B[a]P. Schmahl et al. (1977) tested B[a]P and two mixtures of carcinogenic and noncarcinogenic PAHs when applied to the backs of mice. The tumor response is shown in Section VI of this document to be predictable on the basis of additivity of doses of the carcinogenic mixture components. Assuming simple similar action (i.e., dose additivity) for the purpose of assessing the cancer risk of PAH mixtures seems plausible.

The potential for interactions (synergism or antagonism) tween individual PAHs is another problem that is not addressed

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by the EPA's use of B[a]P as a surrogate for the carcinogenic components of PAH mixtures. The agents in any complex mixture can interact to affect carcinogenesis in a variety of ways. Chemical interactions between agents may create different carcinogenic agents. An example of this in drinking water is the interaction of chlorine used as a bactericide with naturally occurring organic matter to form trihalomethanes (Bellar et al. 1974, Rook 1974). New compounds may form within the body as well. For example, nitrosation of certain compounds in fava beans by endogenous nitrite, when both are present in the gastric lumina, leads to the formation of a potent, direct-acting mutagenic nitroso compound (Yang et al. 1984).

Complex mixtures can also act to modify the exposed individual so that the dose at the site of action for one agent is dependent upon the exposure levels of the other agents in the mixture. Any event that affects the absorption, distribution, metabolism, or elimination of a compound will affect the level of that compound that is available to react with DNA or other target molecules. For example, cigarette smoke can induce the levels of enzymes that metabolize PAHs (Conney et al. 1977), resulting in higher intracellular levels of reactive derivatives capable of modifying DNA.

All such chemical-biological interactions are the result of reactions at many cellular sites with multiple molecules of agents and are probably unimportant at low doses. Mathematical models of the cancer response that depend upon such interaction mechanisms would be expected to be nonlinear at low doses. For

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example, if two chemicals combined to form a carcinogenic agent, the rate of formation would be proportional to the product of the concentrations of the two chemicals. A linear reduction in the concentrations of the chemicals would thus result in a quadratic reduction in the formation of the carcinogenic agent.

The nonlinearity of the typical chemical-biological interaction strongly suggests that mechanisms of carcinogenicity that depend upon such interactions are only marginally important at exposure levels several orders of magnitude below observed responses. Interaction terms that dominate a carcinogenic response at high exposure levels become relatively insignificant at low exposure levels. Even so, any information about chemical interactions or exposure modification should be used in the formulation of a model of the combined effects of agents, if available, by estimating exposure at the cellular and molecular levels.

Interactions may play a large part in carcinogenesis resulting from experimental exposure to PAHs. Mahlum et al. (1984) have shown that different temperature range distillates of coal liquids have different skin tumor-initiating activities in mice despite the fact that they contain similar levels of known carcinogenic PAHs. This difference is believed to be due to the modifying effects of the spectrum of noncarcinogenic PAHs obtained at different temperatures. Most of the PAH components of coal liquid fractions obtained at different temperatures will vary both qualitatively and quantitatively and, as a result, their abilities to modify carcinogenesis will vary accordingly.

This report will examine the biological basis for the mechanisms of action of carcinogenic PAHs and use what is known about such mechanisms to develop a mathematical dose-response model to predict cancer risk resulting from exposure to mixtures of PAHs in the environment. The model is linear at low doses because the mechanisms of interaction that affect tumor rates at the high doses used in the laboratory are hypothesized not to operate at the low doses to which humans are exposed in the environment. This method uses a relative potency approach to account for differing potencies of PAHs as compared to B[a]P, assumes dose additivity, and has fewer limitations than that currently employed.

II. BIOLOGICAL MECHANISMS OF ACTION OF POLYCYCLIC AROMATIC HYDROCARBONS

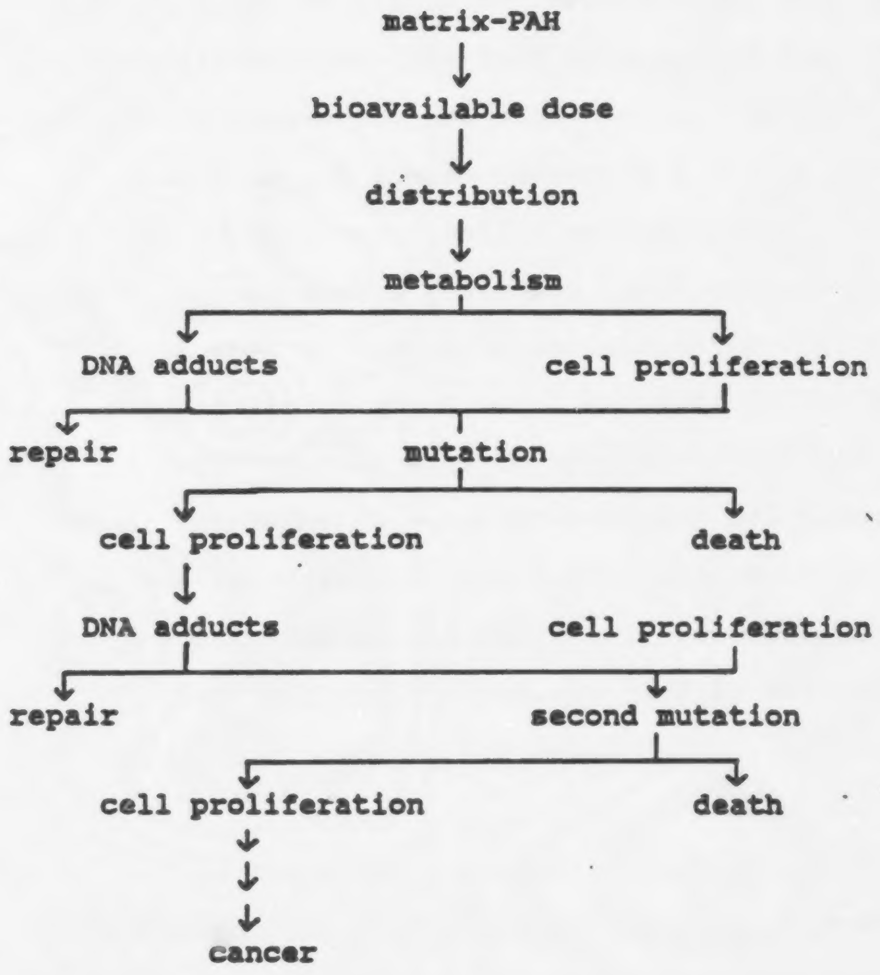
This chapter describes the postulated biological mechanisms of action of PAHs that cause cancer. A series of steps appears to be involved between exposure to PAHs and tumor development; these steps are described below. Each step is expected to occur in a similar manner but to a different extent for different PAHs, which is why their potencies differ. The form of the mathematical dose-response model described in the next chapter depends on the biological mechanisms of action of PAHs.

This section is not intended to be a review of the vast literature available on PAHs but to provide an underlying theoretical rationale for the dose-response relationship to be used for B[a]P-induced cancer risk. Also discussed is how the information that is available for B[a]P may be extended to other PAHs. The organization of this section will follow that outlined in Figure II-1, which shows the proposed basis for PAH-induced carcinogenesis.

The paradigm for PAH-induced carcinogenesis can be summarized as follows. PAHs can be bound to a soil matrix at a hazardous waste site and upon human exposure, a certain fraction of the soil-bound PAHs is released and available for absorption into the body. PAHs are readily distributed throughout the body and are metabolized enzymatically to a number of water-soluble derivatives. A small fraction of some PAHs, if it is not conjugated or deactivated quickly, can be metabolized to a

FIGURE II-1

BIOLOGICAL PARADIGM FOR PAH-INDUCED CARCINOGENESIS


matrix-PAH
bioavailable dose
distribution
metabolism
DNA adducts
cell proliferation
repair
mutation
cell proliferation
death
DNA adducts
cell proliferation
repair
second mutation
cell proliferation
death
cancer

reactive form that may interact with cellular DNA. Once formed, DNA-PAH adducts may be repaired, or if cell proliferation occurs prior to repair, a mutation may occur. The mutated cell may die as a result of the mutation, or may survive and undergo further proliferation and mutation, which can under certain circumstances result in the formation of a malignant tumor. The rest of this section describes each of these steps in greater detail.

1. PAH Matrix and Bioavailabilty

Studies of many complex mixtures have shown that the toxicity of a mixture as it occurs in the environment seldom approaches the theoretical toxicity of the sum of the individual components based on experiments in which pure chemicals are administered individually (Vostal 1983, Umbreit et al. 1986). This finding suggests either that some of the components, or some portions of each component, are not absorbed or that some antagonism among the components occurs.

Bioavailability can be defined as the fraction of a compound in a matrix that is released from that matrix, absorbed by an organism, and, hence, is available to elicit a biological effect. The release and uptake of a compound from a matrix constitute facets of the bioavailability of that compound, although its biological effect is often used as the measure of its bioavailability. Since risk is considered to be a function of both exposure and toxicity, bioavailability is an important consideration in order to determine potential risk from

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components of a complex mixture. The relative proportions of mixture components that reach a tissue are not necessarily the same as that found in the original mixture, and can therefore not be predicted solely by chemical analysis of a mixture.

Little work has been done to determine the bioavailability of PAHs from soil. One study showed that when B[a]P was adsorbed to carbon particles and instilled into the lungs of mice, although clearance of the particle-bound B[a]P was slower than that of unbound B[a]P, less than 15% of the particle-bound B[a]P was eluted from the particles and available to react with respiratory tissue (Creasia et al. 1976).

In another study, Vostal (1983) noted that the in vitro mutagenic activity of diesel particulates, of which PAHs are a major component, was minimal or negative when tested in extracts obtained using biological fluids; furthermore, chronic inhalation exposure of animals to high concentrations of diesel particles did not induce the activity of hydrocarbon-metabolizing enzymes or adversely affect immune response unless organic solvent extracts of the particles were administered intratracheally or parenterally at very high doses. The lack of biological response to diesel particulate matter in the absence of radical treatment with solvents to extract PAHs indicates that environmental exposure to humans could result in a much smaller toxic effect than would be predicted due to limited availability. In some cases, however, gradual leaching of PAHs from retained particles has been shown to lead to an enhanced tumor response as compared to that obtained with bolus dosing.

Studies of the bioavailability from soil of 2,3,7,8-TCDD, a toxic chlorinated polycyclic compound with some physical characteristics in common with PAHS, have shown that bioavailability is a function of soil type, varying from 30% to less than 1% (Umbreit et al. 1986). If PAHs are tightly bound to their soil matrix, as is suspected to be the case at hazardous waste sites such as former manufactured gas plants, the actual exposure to PAHs at the sites will be much less than predicted from simple exposure models because of restricted bioavailability. Moreover, differences in physical properties among the components of PAH mixtures are expected to result in differential bioavailability, compounds of higher molecular weight being more strongly bound to soil than those of lower molecular weight. Differential bioavailability may affect relative potencies. Bioavailability cannot be incorporated into the dose response model for PAHs at this time, however, due to the paucity of data.

2. Distribution

The distribution of many PAHs throughout the body following absorption appears to occur widely and readily. Wide distribution of B[a]P to a variety of body tissues can occur following administration by various routes, and the pattern of distribution is generally similar for all routes of exposure (except for high local pulmonary concentration following intratracheal instillation) (Kotin et al. 1959). IARC (1983) stated

> PAHs are essentially devoid of polar and ionizable functional groups and would therefore be expected to dissolve readily in and cross the lipoprotein membranes of mammalian cells. The demonstrated toxicity of many PAHs in organs remote from the site of their administration confirms this expectation. Furthermore, the fact that isolated cells and tissues metabolize PAHs by means of intracellular enzymes, and that some of these metabolites react with intracellular constituents suggests that uptake across cellular membranes is an easy process.
>
> . . . . Levels of PAHs observed in any particular tissue are dependent on the PAH administered, the route and vehicle of administration, the post-administration sampling times and the presence of inducers of PAH metabolism. Nevertheless, results of a number of studies (Heidelberger & Weiss, 1951; Kotin et al., 1959; Bock & Dao, 1961; Mitchell, 1982) indicate that (i) detectable levels of hydrocarbon can be observed in most internal organs from minutes to hours after administration; (ii) mammary and other fat tissues are significant storage depots where hydrocarbons may accumulate and be slowly released; and (iii) the gut contains relatively high levels of hydrocarbons or hydrocarbon metabolites as the result of hepatobiliary excretion of metabolites or of swallowing of unmetabolized hydrocarbon following mucociliary clearance after inhalation exposure.

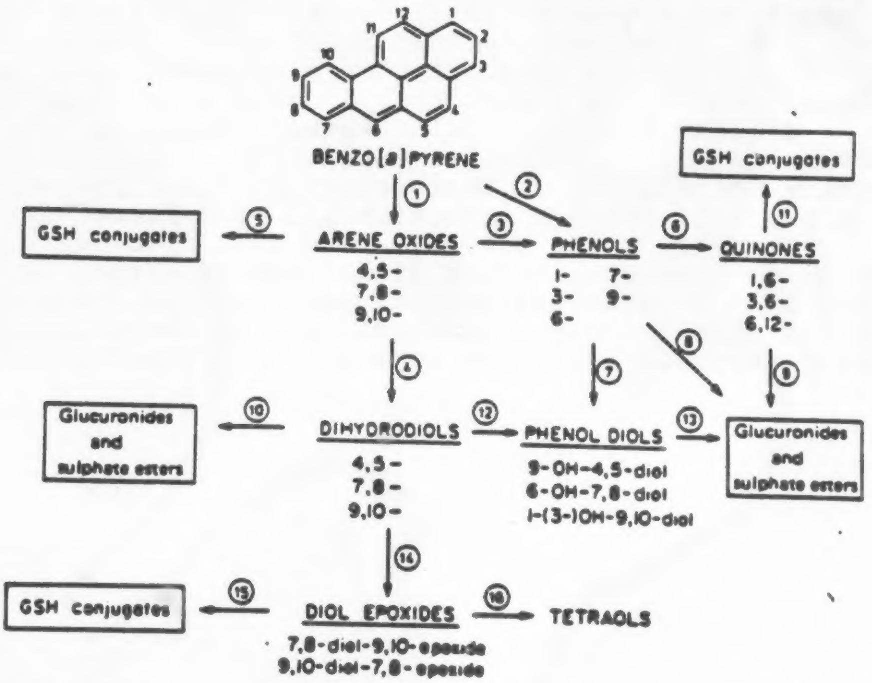
Distribution of PAHs to target tissues thus appears to occur readily (IARC 1983). For the purposes of relative potency estimates and risk assessment, the distribution (equilibrium partitioning) of PAHs will be assumed to resemble that of B[a]P.

3. Metabolism

The metabolism and activation of PAHs have been well reviewed by several authors (Sims and Grover 1974, Conney 1982, Cooper et al. 1983). The general scheme for PAH metabolism is depicted in Figure II-2 for B[a]P. This figure illustrates the multitude and complexity of the metabolic reactions and

FIGURE II-2

METABOLIC FATE OF BENZO[A]PYRENE (FROM IARC 1983)



BENZO[a]PYRENE
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12
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③
④
⑤
⑥
⑦
⑧
⑨
⑩
⑪
⑫
⑬
⑭
⑮
⑯
GSH conjugates
ARENE OXIDES
4,5-
7,8-
9,10-
PHENOLS
1- 7-
3- 9-
6-
QUINONES
1,6-
3,6-
6,12-
GSH conjugates
Glucuronides and sulphate esters
DIHYDRODIOLS
4,5-
7,8-
9,10-
PHENOL DIOLS
9-OH-4,5-diol
6-OH-7,8-diol
1-(3-)OH-9,10-diol
Glucuronides and sulphate esters
GSH conjugates
DIOL EPOXIDES
7,8-diol-9,10-epoxide
9,10-diol-7,8-epoxide
TETRAOLS

Benzo[a]pyrene is metabolized initially by the microsomal cytochrome P-450 monoxygenase system to several arene oxides (reaction 1, Fig. 2). Once formed, these arene oxides may rearrange spontaneously to phenols (reaction 3), undergo hydration to the corresponding trans-dihydrodiols in a reaction catalysed by microsomal epoxide hydrolase (reaction 4), or react covalently with glutathione, either spontaneously or in a reaction catalyzed by cytosolic glutathione S-transferases (reaction 5). Phenols may also be formed by the cytochrome P-450 monooxygenase system by direct oxygen insertion (reaction 2), although unequivocal proof for this mechanism is lacking. 6-Hydroxybenzo[a]pyrene is further oxidized either spontaneously or metabolically to the 1,6-, 3,6-, or 6,12-quinones (reaction 6), and this phenol is also a presumed intermediate in the oxidation of benzo[a]pyrene to the three quinones catalysed by prostaglandin endoperoxide synthetase (Marnett et al. 1977, 1979). Evidence exists for the further oxidative metabolism of two additional phenols; 3-hydroxybenzo[a]pyrene is metabolized to the 3,6-quinone (reaction 6), and 9-hyrdoxybenzo[a]pyrene is oxidized to the K-region 4,5-oxide, which is hydrated to the corresponding 4,5-dihydrodiol (reaction 7). The phenols, quinones and dihydrodiols can all be conjugated to glucuronides and sulphate esters (reactions 8-10), and the quinones also form glutathione conjugates (reaction 11).

FIGURE II-2 (Continued)

METABOLIC FATE OF BENZO[A]PYRENE (FROM IARC 1983)

In addition to being conjugated, the dihydrodiols undergo further oxidative metabolism. The cytochrome P-450 monooxygenase system metabolizes benzo[a]pyrene 4,5-dihydrodiol to a number of uncharacterized metabolites, while the 9,10-dihydrodiol is metabolized predominantly to its 1- and/or 3-phenol derivative (reaction 12), with only minor quantities of a 9,10-diol, 7,8-epoxide being formed (reaction 14). In contrast to 9,10-dihydrodiol metabolism, the principal route of oxidative metabolism of benzo[a]pyrene 7,8-dihydrodiol is to a 7,8-diol, 9,10-epoxide (reaction 14), and phenol-diol formation is a relatively minor pathway. The diol epoxides can be conjugated with glutathione either spontaneously or by a glutathione S-transferase-catalysed reaction (reaction 15). They may also hydrolyze spontaneously to tetraols (reaction 16, although epoxide hydrolase does not catalyze the hydration).

transformations that PAHs may undergo. The majority of PAH metabolites are conjugated as water-soluble derivatives, which facilitates elimination. Some fraction of PAH metabolism may lead to the formation of reactive diol epoxides. The PAH diol epoxide metabolites are thought to be primarily responsible for the ultimate carcinogenic activity of the biologically active PAHs following reactions with critical sites on DNA (Sims et al. 1974). The formation of diol epoxides from B[a]P is outlined in Figure II-3. This figure illustrates that four optically active 7,8-diol-9,10-epoxides are formed from B[a]P. These stereoisomers differ in their rate of formation and in biological activity; the metabolically predominant stereoisomer in rats is also that which possesses the greatest carcinogenic potency (Levin et al. 1982).

Metabolism of PAHs in humans is qualitatively similar to that in a variety of animal tissues, however, quantitative differences are found both among species and among different human tissues and may be influenced by dose and the presence of other PAHs; the susceptibility of a tissue to PAH carcinogenesis may be partly a result of its ability to activate and detoxify PAHs. The potency of PAHs will clearly be related to the extent to which they are metabolized to diol epoxides, the rate at which the diol epoxides are themselves metabolized, and the extent to which those that are formed are capable of interacting with DNA at critical sites.

4. DNA Adduct Formation

The term "DNA adduct" refers to a complex between part of a DNA molecule and a molecule of xenobiotic (or a metabolite). Covalent binding of reactive metabolites of B[a]P and other carcinogenic PAHs to DNA can occur and results in the formation of DNA adducts. This step appears to be essential for the production of PAH-induced neoplasia (Gelboin 1980, Weinstein et al. 1978). If the adducts are not repaired, replication of damaged DNA during cell proliferation may lead to mutation. The production of neoplasia may be related both to the extent to which PAHs bind to DNA, and the level of cell proliferation in the target tissue.

Pereira et al. (1979) applied single doses of B[a]P topically to ICR/Ha mice and found a linear relationship between the applied dose and the amount of each of the two major B[a]P-DNA adducts formed in the epidermis over a dose range of 0.01 to 300 mg B[a]P per mouse. The level of adduct formed reached a maximum at 7 hours and remained constant for the next 49 hours, indicating that repair of such lesions is slow.

Adriaenssens et al. (1983) administered single doses of B[a]P orally via intubation to rats and examined the formation of DNA adducts over a dose range of 2 to 1,351 umol/kg (0.048 to 29.7 umol/mouse). The rate of formation of the major diol epoxide-DNA adduct was linear with respect to dose in the forestomach and, although the authors stated otherwise, in the lung as well. The extent of the deviation from linearity in the

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lung was slight and consistent with the level of error associated with this type of measurement.

PAH metabolites have been found to bind to DNA in every tissue that has been examined, regardless of species, dose, or route of administration. The physical nature of the adducts formed, the levels at which they occur, and the extent to which they persist unrepaired appear to be similar in various tissues, whether or not those tissues are likely to develop tumors (Stowers and Anderson 1985). Thus, formation of PAH-DNA adducts appears to be a necessary, but not sufficient step for PAH-induced carcinogenesis; another step is also required.

5. Cell Proliferation

The rate of cell proliferation can have an important influence on the rate at which mutation occurs and may account for the differences in susceptibility among tissues. Experiments with radiation and chemically-induced mutagenesis have indicated that at least one round of cell proliferation is required before the mutational change becomes permanent and cannot be repaired (Borek and Sachs 1968, Kalanuga 1974). Increasing the rate of cell proliferation in a tissue can increase the susceptibility of that tissue to mutation (Craddock 1971). Cells that are replicating are known to be of greater susceptibility to mutation than those that are not (Tong et al. 1980). Cells that are not actively proliferating have time to repair DNA damage, making the occurrence of a mutation much less likely.

The role of cell proliferation in PAH-induced carcinogenesis is two-fold:

(1) Tissues that have higher rates of cell turnover, such as the skin and lung, appear to be target tissues for tumor formation, while those with slower rates, such as the liver, are not. For example, B[a]P does not usually cause liver tumors in rats. Performing partial hepatectomy on rats leads to a high rate of hepatic cell turnover. If partial hepatectomy is performed prior to B[a]P administration, tumors will occur in that organ (Hirakawa et al. 1979).

(2) PAH administration can increase cell proliferation as a consequence of toxicity. The formation of bulky DNA adducts and adducts with other cellular macromolecules such as RNA and protein can lead to toxicity and cell death. One response of a tissue to toxicity is to increase cell proliferation in order to replace damaged cells. Epidermal hyperplasia has been shown to occur following topical application of B[a]P and its diol epoxide to the backs of C57BL/6J mice (Bresnick et al. 1977). A linear dose-response relationship occurred between the number of layers of epidermis following a single dose of the carcinogenic diol epoxide of B[a]P over the dose range of 0.2 to 1.2 umol/mouse; the increase persisted for 4 days. These observations indicate that, at least at high doses, B[a]P and its metabolites may not only form DNA adducts, but may increase

III. DEVELOPMENT OF A COMPARATIVE POTENCY APPROACH FOR THE ASSESSMENT OF CANCER RISK ASSOCIATED WITH MIXTURES OF POLYCYCLIC AROMATIC HYDROCARBONS

This chapter describes the mathematical models that could be used to characterize the dose response relationships between PAHs and cancer using laboratory animals and thereby to predict the human cancer risk associated with exposure to mixtures of PAHs. The models are derived, put in a biological context, and their strength and weaknesses noted. A general model is recommended and made specific for B[a]P using data from experiments in which mice were fed or hamsters inhaled B[a]P and their tumor responses were determined. The assumptions that are required to convert a dose response model based on animal experiments into one that predicts lifetime human cancer risk, if levels of human exposure are known, are described and used to obtain a human dose response model for B[a]P. Finally, a method is derived that uses this model in conjunction with relative potency estimates for other PAHs to estimate the human cancer risk associated with exposure to mixtures of PAHs.

1. Classic Multistage Model

Armitage and Doll (1954) derived a mathematical model for carcinogenesis that assumes a complex, time-dependent process involving a specific ordering of at least two qualitatively different cell stages. Under the assumption that the transition rate (r_i) between the i-1 and ith cell stages is constant over time ($i = 1,2,...k$) and that a total of k stages

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must occur to produce a neoplastic cell, the probability that a tumor will occur by age t in the absence of competing risk is

$$P(t) = 1 - e^{-N \left(\prod_{i=1}^{k} r_i\right) t^k/k!} \qquad \text{(III-1)}$$

In this formulation, it is assumed that each of the N susceptible cells in the target organ is independently undergoing transition to its next stage. It is also assumed that one or more of the transition rates is a linear function of the exposure level x. Using the model parameters, this is equivalent to specifying

$$r_i = a_i + b_i x \qquad i = 1,2,\ldots,k,$$

where $a_i > 0$ and $b_i \geq 0$. The latter condition excludes the possibility that the carcinogen may be inhibitory (i.e., reduce the transition rate) at any stage. The number of stages, m, that have transition rates that are exposure-dependent defines the degree of the polynomial relationship. The exponent of the multistage model in this form is a constant times the product of m linear equations with non-negative coefficients. In the resulting polynomial, each of the coefficients associated with terms of order m or less exist and are subject to certain nonlinear constraints. For example, if the coefficient corresponding to the jth power is denoted as C_j and two stages are exposure-dependent (i.e., m = 2), it follows that $C_j > 0$ for $j = 0,1,2$ and $C_1 \geq 2(C_0C_2)^{1/2}$ (see Kalbfleish et al. 1983).

It is noteworthy that this form of the multistage model has seldom been used in risk assessments but is usually referred to as the main rationale for low-dose linearity. Its advantages are that it stems from a reasonable biological theory of carcinogenesis and yields a positive, nonzero maximum likelihood estimate of the linear term. Its disadvantages are that the nonlinear constraints on the C_i's often create estimation problems and that dose-response relationships with high upward curvature could not be fitted with this model.

2. Linearized Multistage Model

To avoid the problems posed by the classic multistage model, Guess and Crump (1977) "generalized" the multistage model by giving it a less restrictive polynomial form:

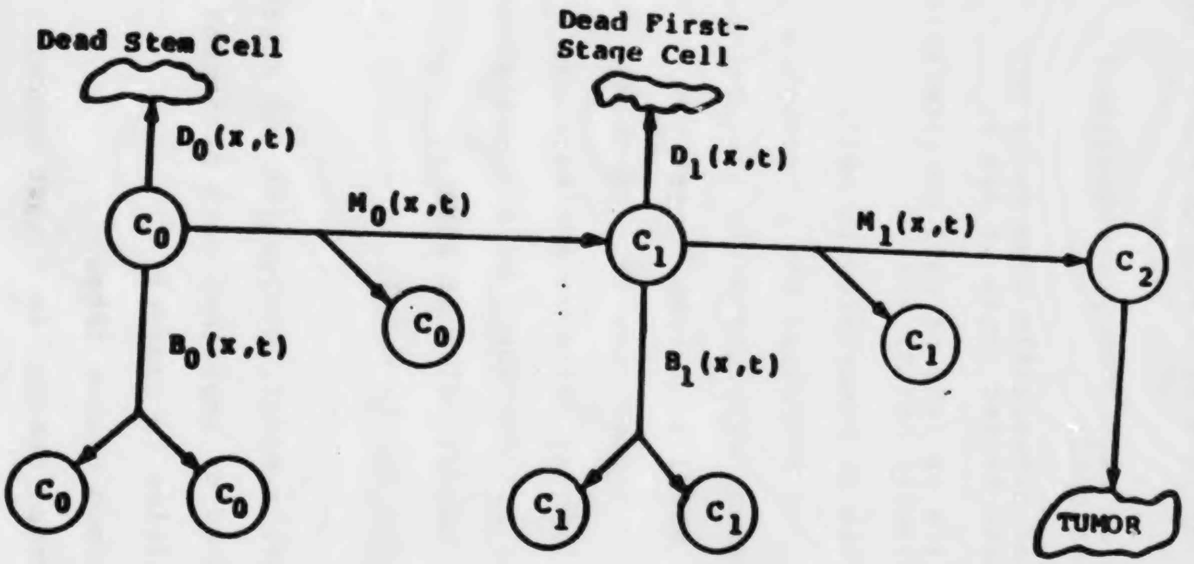
$$P(x,t) = 1 - e^{-\left[\sum_{j=0}^{m} c_j x^j\right] t^k} \qquad \text{(III-2)}$$

where $C_j \geq 0$ for $j = 0,1,\ldots,m$. When the model is fitted to data from an experiment with (n+1) groups of animals (usually one control group and n dose levels), the constraint $m \leq n$ is applied.

As a first step when using the model, the maximum likelihood estimates for the C's are obtained. Next, a unique polynomial is derived that gives the maximum risk at a specified level of exposure, subject to a goodness-of-fit constraint. The linear term in this polynomial times an model is depicted in Figure III-1. According to the model, the population of cells at risk is proliferating cells, often referred to as stem cells. Stem cells are those from which most other cells in an organ arise. A stem cell can differentiate and leave the pool of proliferating cells, becoming no longer susceptible to heritable alterations of its DNA. A normal, susceptible stem cell may do one of several things. It may divide into two daughter stem cells, terminally differentiate, die, or undergo mutation at a critical site that results in formation of a preneoplastic or intermediate cell. A preneoplastic cell may be defined as a cell that has undergone one of the changes necessary to become a cancer cell but is not yet cancerous. The preneoplastic cell may, in turn, divide into two daughter preneoplastic cells, differentiate, die, or undergo further mutation at another critical site to produce a cancerous cell. The cancerous cell will, after a sufficient length of time, divide into enough cells that it becomes a detectable tumor. All of these processes can be described mathematically by postulating specific exposure-dependent rates for the cell changes. Moolgavkar and Knudson (1981) showed that to a close approximation, the age-specific tumor rate at time t for their two-stage model may be expressed as:

$$I(t) = M_0 M_1 \int_0^t C_0(v) \, [\exp (B-D)(t-v)] \, dv \qquad \text{(III-3)}$$

FIGURE III-1
GENERAL BIOLOGICAL STRUCTURE UNDERLYING EXPOSURE- AND TIME-DEPENDENT MODEL BASED ON THAT DESCRIBED BY MOOLGAVKAR, VENZON, AND KNUDSON


Dead Stem Cell
$D_0(x,t)$
C_0
$M_0(x,t)$
C_0
$B_0(x,t)$
C_0
C_0
Dead First-Stage Cell
$D_1(x,t)$
C_1
$M_1(x,t)$
C_1
$B_1(x,t)$
C_1
C_1
C_2
TUMOR

KEY:

C_0 is a normal, susceptible stem cell.

C_1 is a transformed, first-stage cell, which can proliferate into a premalignant clone.

C_2 is a cancerous cell that will eventually develop into a detectable tumor.

$D_0(x,t)$, $B_0(x,t)$, and $M_0(x,t)$ are the exposure- and time-dependent death, birth, and transition or mutation rates for the normal stem cell.

$D_1(x,t)$, $B_1(x,t)$, and $M_1(x,t)$ are the exposure- and time-dependent death, birth, and transition or mutation rates for the first-stage cell.

x is the exposure level, which is assumed to be constant over time.

t is the age of the subject.

where

$I(t)$ = age-specific cancer incidence at age t;

M_0 = transition rate from stem to preneoplastic cell;

M_1 = transition rate from preneoplastic to cancerous cell;

$C_0(v)$ = number of susceptible stem cells per individual target organ at age v;

B = birth rate or rate of cell proliferation of preneoplastic cells; and

D = death rate of preneoplastic cells.

Essentially, the equation describes the progression from a normal stem cell to a cancerous cell under the assumptions of the two-stage model. This model is a combination of deterministic and stochastic components. The numbers of preneoplastic and fully malignant cells at any time are assumed to be random variables that are dependent upon these event rates, while the number of normal cells at risk of transformation at time v, denoted by $C_0(v)$, is assumed to be deterministic and known.

Using this carcinogenesis model, Moolgavkar and Knudson (1981) successfully described or explained such phenomena as

- genetic predisposition to cancer;
- patterns in childhood cancer rates;
- hormonally influenced changes in breast cancer rates;
- the results of initiation-promotion experiments for multiple agents;
- changes in respiratory cancer rates associated with variable smoking patterns; and

- the fact that for many human carcinomas, the age-specific incidence rates increase roughly with the fourth to the seventh power of age.

The Moolgavkar-Knudson model is thus more general in applicability than the Armitage-Doll model, which was developed specifically for carcinomas in epithelial cells and predicts an age-specific incidence approximately proportional to t^{k-1}, where k is the assumed number of stages.

The biological processes described by equation III-3 can be affected by the level of exposure to carcinogenic agents, and are more likely to occur as the length of exposure increases; as a result, they are exposure and time dependent. Thorslund et al. (1987) have described an exposure- and time-dependent version of the model. In the context of biological mechanisms of carcinogenesis, the model can be used to predict the risk of agents that exert their effects in a number of different ways. Mutation-inducing initiating agents would affect the transition rates between cell stages (M_0 or M_1), while promoting agents may increase the proliferation rate of preneoplastic cells (increasing B without affecting D). Cocarcinogens may increase the proliferation rate of normal stem cells (C_0), thereby increasing the size of the target for initiating agents. Inhibitors could remove cells from the populations of susceptible stem or preneoplastic cells through toxicity (e.g., increasing D without affecting B) or by inducing differentiation. Within the context of the model, the following observations can be explained:

- tumor rates at very high experimental doses are often lower than at lower doses, even after adjusting for differential mortality, as a result of high-dose toxicity to stem and preneoplastic cells;
- exposure to multiple carcinogens can give antagonistic, additive, multiplicative, or super-multiplicative tumor rate responses by affecting different model parameters;
- such a response to combined agents appears to be dose-dependent because some responses such as increased stem cell proliferation may both occur and have an impact on tumor rates only at high doses; and
- some preneoplastic, benign cell masses that may be precursors of tumors will regress and disappear upon cessation of exposure as a result of a decline in the birth rate and no change in the death rate of such cells.

The multistage model provides no satisfactory explanation for any of these observations.

4. Restricted Dose Response Model for B[a]P

B[a]P is a carcinogenic polycyclic aromatic hydrocarbon that occurs ubiquitously in air and soil as a product of combustion. B[a]P can be metabolically activated to form derivatives that can react with DNA (Sims and Grover 1974, Gelboin 1980, Weinstein et al. 1984). If B[a]P-DNA adducts occur at critical sites that control the regulation of a cell's growth or differentiation, mutations may occur that can lead to cell transformation and ultimately, cancer. Because the experimental evidence (Lee and O'Neil 1971) strongly suggests that B[a]P is a genotoxic agent acting at at least two sites on DNA, the two-stage model is a useful approach for estimating the cancer risk associated with exposure to B[a]P. Without intermediary experimental information about cell stages and differences in exposure over time, however, it is not possible to estimate the individual background and exposure-induced mutation rates for preneoplastic and transformed cells using tumor rate data from a standard animal carcinogenesis bioassay, nor can the relative transition rates that correspond to each stage be identified. What can be estimated from bioassay data are two exposure-induced relative transition rates and a background transition rate. In the absence of information to the contrary, the assumption was made that the transition rates from normal to preneoplastic and preneoplastic to cancerous cells are equally likely, which could occur if the mutations that caused the transformations resulted from the same type of DNA adduct. If these transition rates are linear functions of dose (which is likely at low doses) and the growth rate of preneoplastic cells is independent of exposure level, the probability that a tumor will develop by time t as a result of exposure to a level of genotoxic agent x can be expressed as:

$$P(x,t) = 1 - \exp - \{M(1+Sx)^2 [\exp(Gt) - 1 - Gt]/G^2\} , \quad \text{(III-4)}$$

where equation III-3 was integrated to obtain the cumulative hazard function and

M = background tumor rate parameter;

S = fractional increase in the transition rate between cell stages per unit dose, assumed to be the same for each stage;

G = the difference between the birth and death rates of preneoplastic cells, B-D, and is the exposure-independent growth rate of these cells; and

t = the time (or age) at which risk is evaluated.

The level of x at the target tissue is assumed to be directly related to the administered dose, based on observations of experiments discussed in Section II in which the rate of formation of the major B[a]P diol epoxide-DNA adduct was found to be linearly related with respect to dose in the forestomach, lung, and skin (Pereira et al. 1979, Adrienssens et al. 1983).

The specific dose-response model derived for B[a]P (equation III-4) is restricted form of the model developed by Armitage and Doll (1957), Moolgavkar and Knudson (1981), and Thorslund et al. (1987). It is restricted by assuming that G is independent of x and that the two transition rates are linear functions of x with the same coefficient. The consequence of these assumptions is that at constant t the dose-response function has only two parameters:

$$P(x) = 1 - \exp{-A(1 + Sx)^2} \qquad \text{(III-5)}$$

where $A = M\,[\exp(Gt)-1-Gt]/G^2$ and t = age at last observation.

This model has a number of advantages. Among the most important are the following:

- At low doses, it converges to a linear, no-threshold form with slope parameter equal to 2AS.
- The model can be fitted to data from an experiment in which only one exposure group exhibited a positive response.
- The model has only two parameters that have to be estimated, so that X^2 goodness-of-fit tests can be run on the data from the standard bioassay with one control and two exposure groups.
- A stable point estimate of risk can be obtained directly.

- The mathematical form of the model follows directly from what is currently the most widely accepted hypothesis of the mechanisms of cancer induction.
- The model is consistent with the mechanisms of tumorigenesis for B[a]P and other PAHs postulated in Section II.

It should be noted that equation (III-5) is also a restricted form of the Armitage-Doll (1954) model, with two stages affected equally by the carcinogen. Under these restrictions, equations (III-1) and (III-4) differ only in their dependence on t. It is not possible to distinguish between the two models on this basis, however, unless an integer power of t is inconsistent with the data. In such a case, the Armitage-Doll model would be rejected.

The assumption that the parameters A and S in equation (III-5) are greater than zero avoids the disadvantages of the linearized multistage model that are listed in Section III.2; hence, equation (III-4) or (III-5) can be used to derive stable point estimates of low-dose risk. Such estimates are derived for B[a]P in the succeeding sections of this chapter, and for other PAHs in Section IV. However, because equation (III-2) includes equation (III-5) as a special case, the Guess-Crump algorithm provides reasonable estimates of an upper bound on low-dose risk for these chemicals. Such estimates are derived in Section VI and are compared with point estimates derived in Sections III and IV, with upper bounds derived from the multistage and point estimates from the one-hit models.

5. Dose-Response Relationship for Oral Exposure to B[a]P

The carcinogenicity of B[a]P via ingestion was investigated in a series of experiments conducted by Neal and Rigdon (1967) and Rigdon and Neal (1966, 1969). The most pronounced dose-response relationship obtained under reasonably consistent conditions was observed in the Neal and Rigdon (1967) study. The results of this experiment are reproduced in Table III-1. The end point in this study was either a papilloma or a squamous cell carcinoma in the forestomach (squamous portion of the stomach), although squamous cell carcinomas were far more common.

A number of experimental factors that are atypical of the standard bioassay protocol were employed in the Neal and Rigdon (1967) study. These included variable age of first exposure, a duration of exposure that only lasted about one-seventh of a lifetime, and an observation period that was less than one-fifth of a lifetime.

Syracuse Research Corporation (SRC), under contract to the U.S. Environmental Protection Agency (EPA), had only limited resources to devote to developing a unit risk estimate for B[a]P. They therefore decided to use the Neal and Rigdon (1967) study but restrict the data set to that subgroup of the experimental population that had been exposed continuously for the length of the experiment (EPA 1984). In fitting the linearized multistage model, no adjustments were made for the variable length of the follow-up period or the age attained at the end of the observation period. Using the standard EPA

TABLE III-1

FORESTOMACH TUMORS IN MICE FED BENZO[a]PYRENE

Age First Exposed (days)	Milligrams of B[a]P per gram of food	Number of days fed B[a]P	Age killed (in days)	Number with Forestomach Tumors / Number of Mice
--	0.0	--	300	0/289
30	0.001	110	140	0/25
30	0.01	110	140	0/24
116	0.02	110	226	0/24
33-67	0.03	110	143-177	1/23
33-101	0.04	110	143-211	0/37
31-71	0.045	110	143-183	1/40
17-22	0.05	107-197	124-219	4/40
20-24	0.10	98-122	118-146	24/34
18-20	0.25	70-165	88-185	19/23
49	0.25	1	155	66/73
56	0.25	2	162	0/10
49	0.25	4	155	1/9
62	0.25	5	168	1/10
49	0.25	7	155	4/9
91	0.25	30	198	3/10
74	0.10	7	182	26/26
48	0.10	30	156	0/10
98-180	5.0	1	209-294	12/18
				17/33

SOURCE: Adapted from Neal and Rigdon (1967) approach discussed in Anderson et al. (1983), SRC discarded all data on animals exposed to 50 ppm B[a]P or more because of "lack of fit" and obtained a 95% upper-bound estimate on the chemical's potency to humans of 11.53 $(\text{mg/kg/day})^{-1}$. This number represents an upper bound on the slope of the cancer dose-response curve for this chemical.

If more detailed data were available on the age at first exposure and the length of exposure for individual animals, rather than for exposure groups as a whole, a more precise, time-adjusted analysis would be possible.[1] However, because such detailed information is lacking, the approach taken here is to restrict the analysis to exposure groups that are comparable with respect to age at first exposure and number of days exposed. The resulting homogeneous experimental subpopulation selected from the entire experimental population (see Table III-1) is presented in Table III-2. This is the same subgroup used by Chu and Chen (1984).

The two-stage model with identical linear dose-dependent transition rates (equation III-5) was fitted to the data in Table III-2. The joint maximum likelihood estimates of the parameters when there are no animals with tumors in the control group can yield estimates where $A = 0$. One way around this problem is to obtain a positive estimate for A using an alternative method. Based on *a priori* biological assumptions, we know that $A > 0$. This follows from the fact that

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[1] Methods could be developed to use more fully the types of data that are available, including the information presented in the other Rigdon and Neal (1966, 1969) papers. However, such an effort is beyond the scope of the current project.

TABLE III-2

DATA USED TO ESTIMATE THE DOSE-RESPONSE RELATIONSHIP BETWEEN INGESTED B[a]P AND FORESTOMACH TUMORS

Dose (ppm in diet)	Dose (x) (mg/kg/day)	Number of Mice with Forestomach Tumors / Number Exposed	Expected Number of Tumors Predicted by Model
0	0.00	0/289	0.50
1	0.13	0/25	0.05
10	1.30	0/24	0.20
30	3.90	0/37	1.32
40	5.20	1/40	2.25
45	5.85	4/40	2.72

NOTE: This table contains data only on those groups that are comparable with respect to age when first exposed and number of days exposed. The first three columns are from Neal and Rigdon (1967). The fourth column is based on the low-dose linear two-stage, dose-dependent, time-independent, identical transition rate model:

$$P(x) = 1 - \exp [-0.0017256(1 + 0.922x)^2]$$

if one assumes that B[a]P exerts its effects via a direct genotoxic mechanism of action, the background tumor rate is proportional to the square of the mutation rate caused by factors other than B[a]P at the critical gene locus. Since a variety of other factors such as background levels of PAHs, viruses, and solar irradiation have the potential to induce such mutations, a nonzero background tumor rate estimate is required for biological consistency. Unfortunately the "maximum likelihood" estimate of the background tumor rate is zero (i.e., 0/289 = 0). As a result, an alternative estimation procedure will be used to estimate the background tumor rate. When the observed background tumor rate is zero, the multistage procedure often gives a zero estimate of the true background rate. Such an estimate is illogical from a biological standpoint, implying that exposure to PAHs is a necessary condition for the occurence of the tumor in question. The two-stage model is applied in this report so as to make a zero background rate estimate impossible. The approach taken to estimate the background rate is to use a Bayesian estimator with an invariant prior. This approach has been suggested by Jeffreys (1961, pp. 117-125) and Gart (1966), among others, in comparable situations where it is known a priori that a zero estimate is unlikely for a binomial response.

Using this approach, the background tumor rate is estimated to be

$$P(0) = 0.5/(n + 1) = 0.5/290 = 0.0017241.$$

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Equating this value to the parametric form of the two-stage, dose-dependent, identical transition rate model yields the following relationship:

$$P(0) = 0.0017241 = 1 - \exp\left[-A(1 + S \cdot 0)^2\right] = 1 - \exp(-A),$$

which may be solved directly for A. The solution is A = 0.0017256, which is put into the model equation to obtain the relationship:

$$P(x) = 1 - \exp\left[-0.0017256(1 + Sx)^2\right].$$

The remaining unknown parameter, S, is then estimated by fitting the above equation in the data in Table III-2. An approximate maximum likelihood estimate of 0.922 is obtained. The goodness of fit of the model is also shown in Table III-2. The expected values are too small to conduct a formal X^2 goodness-of-fit test. However, it is apparent that the model does not underestimate the tumor rate or overestimate the curvature in the experimental range. The data are thus consistent with the restricted two-stage model represented by equation III-5.

The low-dose linear term from this model may be expressed in terms of the parameter values as 2AS, which must be greater than zero because the background rate A is always greater than zero and S is greater than zero for all positive dose-response

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relationships. The low-dose linear term in animal exposure units (mg/kg/day) for a 140-day (30+110) experiment is thus

$$2AS = q_1 = (2)(0.001723)(0.922) = 0.00318\ (mg/kg/day)^{-1}.$$

To translate this into a human potency value, two adjustments are necessary. First, the risk must be adjusted to account for constant exposure throughout one's entire lifetime. It is assumed that 2 mice years equal 70 human years (i.e., a lifetime) and (2 mice years)(365 days/year) = 730 days. EPA's standard procedures for less than full lifetime follow-up yield an adjustment factor of $(730/140)^3 = 141.8$ (Anderson et al. 1983). Second, it is standard practice to calculate species-equivalent exposure units on a mg/surface area basis. To convert the estimate to these units, it is multiplied by the additional adjustment factor of $(70/0.034)^{1/3} = 12.72$, where 70 and 0.034 kg are the weights of an average human and CFW mouse, respectively. After making these two adjustments, the human potency factor is estimated to be

$$q_1 = (0.00318)(141.8)(12.72) = 5.74\ (mg/kg/day)^{-1}$$

for human exposure. This value can be compared to the 95% upper-bound estimate of 11.53 $(mg/kg/day)^{-1}$ obtained by EPA's contractor, which is not an actual estimate; it is only the upper bound on the actual values and is approximately two times larger than the linear point estimate obtained here.

6. Dose-Response Relationship for Inhalation Exposure to B[a]P

The preferred route of exposure for chronic studies conducted to assess the risk of cancer posed by airborne B[a]P is inhalation. However, such studies have difficult methodological problems associated with them. As a result, much of the work on this chemical has been done using intratracheal instillation of B[a]P alone or in combination with other agents as a surrogate route of exposure for inhalation. Although much valuable qualitative information has been obtained by this approach, its use in quantitative risk assessments has not met with much success.

Most carcinogenesis bioassays in which B[a]P has been administered via inhalation exposure have yielded negative results. There are several exceptions; most notably, a positive response was obtained in rats using a combination of B[a]P and the atmospheric irritant sulfur dioxide; sulfur dioxide by itself was not carcinogenic (Laskin et al. 1970). The tumors induced in this experiment were squamous cell carcinomas; a proportion of the bronchogenic carcinomas found in humans are of this type.

A well-conducted study by Thyssen et al. (1981) provides the most clear-cut evidence of the dose-response relationship between inhaled B[a]P and tumorigenesis. In this experiment, Syrian golden hamsters were exposed throughout their lives to B[a]P in a sodium chloride aerosol for 4.5 hours per day, 7 days per week for 10 weeks and then 3 hours per day thereafter. Respiratory tract tumors were induced in the nasal

TABLE III-3

DATA USED TO ESTIMATE DOSE-RESPONSE RELATIONSHIP BETWEEN INHALED B[a]P AND RESPIRATORY TRACT TUMORS

Exposure Rate (x) (mg B[a]P/m^3) Observed	Average Survival (t)	Effective Number (in weeks)	Number of Respiratory Tract Tumors	
			Exposed	Predicted
0	96.4	27	0.73	0
2.2	95.2	27	1.88	0
9.5	96.4	26	9.06	9
46.5	59.5	25	12.59	13

NOTE: Based on low-dose linear two-stage, dose- and time-dependent, identical transition rate model:

$$P(x,t) = 1\text{-exp } [-0.000115(1+0.312x)^2][\exp(0.057t)-1-0.057t]$$

SOURCE: Thyssen et al. 1981

TABLE III-3

DATA USED TO ESTIMATE DOSE-RESPONSE RELATIONSHIP BETWEEN INHALED B[a]P AND RESPIRATORY TRACT TUMORS

Exposure Rate (x) (mg B[a]P/m^3) Observed	Average Survival (t)	Effective Number (in weeks)	Number of Respiratory Tract Tumors	
			Exposed	Predicted
0	96.4	27	0.73	0
2.2	95.2	27	1.88	0
9.5	96.4	26	9.06	9
46.5	59.5	25	12.59	13

NOTE: Based on low-dose linear two-stage, dose- and time-dependent, identical transition rate model:

$$P(x,t) = 1-\exp\,[-0.000115(1+0.312x)^2][\exp(0.057t)-1-0.057t]$$

SOURCE: Thyssen et al. 1981

To extrapolate this value to human exposure for 24 hours per day, the average length of exposure per day over the approximate 2-year experimental period is calculated. The weighted mean of the two exposure periods is $[(10)(4.5)+(92)(3)]/102 = 3.147$ hr/day. Thus, exposure 24 hours per day has a potency value of

$$q_1 = (0.0170)(24/3.147) = 0.1295\ (\mathrm{mg/m^3})^{-1}.$$

This calculation is based on the assumption that inhalation exposures are equivalent across species. To convert this exposure level to $(\mathrm{mg/kg/day})^{-1}$ to be able to compare it to exposure via ingestion, the absorption rates for inhalation and ingestion are assumed to be the same and the average person is assumed to weigh 70 kg and inhale 20 m^3 air per day. Under these assumptions, exposure to 1 $\mathrm{mg/m^3}$ for 24 hours results in an exposure level of $[(1)(20)]/70 = 0.2857$ mg/kg/day. Thus, on a mg/kg/day basis the estimated slope is

$$q_1 = 0.1295/0.2857 = 0.4533\ (\mathrm{mg/kg/day})^{-1}.$$

This value is considerably smaller than the EPA's estimate of 6.11 $(\mathrm{mg/kg/day})^{-1}$, which is based upon a species adjustment for metabolism that corrects for the differences in metabolic rates in an unclear manner.

The data of Thyssen et al. (1981) can also be fitted to the Armitage-Doll model (equation III-1), with the assumption

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that two stages are equally affected by the carcinogen out of a total of 3 or 4 stages. Thus, both models can be fitted to the data; the conceptual difference between the two models is that the Moolgavkar-Knudson model is limited to two stages with well-defined biological transitions between them, whereas the Armitage-Doll model requires one or two additional stages and does not specify the nature of the transitions.

7. Estimation of the Joint Carcinogenic Response to Total PAH Exposure

In this section, a method for obtaining relative carcinogenic potency estimates for PAHs is derived. This method is dependent upon the structural form of the dose-response model developed for B[a]P. Derivation of relative potency estimates requires that point estimates of risk be obtained because upper bounds on risk cannot be compared. For these reasons, point estimates of risk were derived by fitting equation III-5 to dose-response data for various PAHs. The point estimates are compared to the corresponding estimates for B[a]P to obtain relative potency estimates. These relative potency estimates serve as the parameters Rj in equation I-1 (presented in Section I).

The two-stage model was shown earlier in this section to be consistent with the dose-response relationship between B[a]P and mouse forestomach tumors following oral administration and hamster lung tumors following inhalation exposure; Lee and O'Neil (1971) have demonstrated a similar relationship for mouse skin tumors following topical application. Given this demonstrated experimental consistency and the underlying theoretical rationale for the mechanism of action of the PAHs (see Section II), it is reasonable to assume that the same functional relationship exists for all animal test models. The parameter estimates would undoubtedly be species- and tissue-dependent, but the underlying structural relationships would very likely be the same.

Using the low-dose linear two-stage identical transition rate model developed for B[a]P, the dose-response relationship for a specified test system can be expressed as

$$P(x) = 1\text{-}\exp[-A(1+Sx)^2]$$

for B[a]P and

$$P(y_j) = 1\text{-}\exp\text{-}A(1 + SR_j y_j)^2 \quad j = 1,2,\ldots,m$$

for the j'th carcinogenic PAH.

It is possible to obtain 100% efficient estimates of the R_j values by joint maximum likelihood estimation using data from bioassays of individual PAHs conducted simultaneously. However such estimation procedures are time-consuming to develop and lie beyond the scope of the present project. Fortunately, a simple approximation for the estimates of the R_j values can be obtained. To do so, the value of A is estimated from the control data as previously discussed. It can be shown that the final potency estimates are not very sensitive to this estimate. Given this estimated value of A, the term SR_j can be estimated by an approximate goodness-of-fit procedure for each PAH for which data are available. The parameter S is estimated in a comparable manner using B[a]P dose-response data from the same bioassay. The final relative potency estimates R_j, which according to our general theoretical approach are independent of animal test system used, are obtained from the ratio of the estimates for SR_j and S, determined from the jth PAH and B[a]P, respectively.

For many test systems, the highest dose may be the least relevant for determining potency due to acute toxicity, activation of cell proliferation mechanisms, and/or saturation of metabolism or local penetration. When the highest dose yields a level that is inconsistent with the assumed dose-response model, it will not be used in estimating the parameters. In other situations, only a single dose level for B[a]P may be available. In this case, if B[a]P gives a high response, a more stable estimate may be obtained by simply using the PAH exposure level that gives the closest response rate to that of B[a]P since such a procedure is independent of any assumed parametric dose-response relationship. Even when a control value and a single response for B[a]P and the jth carcinogenic PAH are the only values available, it is possible to obtain an estimate of the relative potency. To demonstrate how such estimates can be obtained, consider the following limited response data:

OBSERVED RATES

Agent	Exposure	Response
Control	0	r_o/n_o
B[a]P	x	r/n
jth PAH	y_j	r_j/n_j

Estimates for the relative potencies can be obtained by equating the observed rate with the function response. This results in the equations

$$r_o/n_o = 1\text{-exp-}A$$

$$r/n = 1\text{-exp-}A(1+Sx)^2$$

$$r_j/n_j = 1\text{-exp-}A(1+SR_jy_j)^2 \quad j = 1,...,m$$

which can be solved algebraically by hierarchical substitution of the parameter estimates for A and S into the equation defining the relative potencies. This approach yields the algebraic solution:

$$R_j = \frac{x}{y_j}\,\frac{[\ln(1-rj/nj)/\ln(1-r_o/n^o)]^{1/2} - 1}{[\ln(1-r/n)/\ln(1-r_o/n_o)]^{1/2} - 1} \qquad j = 1,2,...,m.$$

In the next section, the relative potency values (R_j) are estimated for selected PAHs that are likely to be encountered in the environment.

IV. THE BASIS FOR AND DERIVATION OF COMPARATIVE POTENCY ESTIMATES

This chapter describes nine experiments in which B[a]P and other PAHs were tested concomitantly for carcinogenesis using several animal species and different methods of administration, as well as two experiments in which the levels of these chemicals that interact with cellular DNA were determined. Unlike the experiments using B[a]P described in Section III, most of these studies used methods of administration that cannot be directly compared to those by which humans would be expected to be exposed. As a result, dose reponse relationships for these PAHs cannot be described mathematically in a manner that is useful for the prediction of human risk unless they are expressed relative to B[a]P. Cancer potencies relative to B[a]P are derived for each PAH from each experiment in the following section. The studies that are considered the best in terms of performance and relevance to humans are then selected; the relative potencies for the PAHs from these studies are summarized at the end.

A. CARCINOGENESIS

It is difficult to compare directly the results of carcinogenesis bioassays because of inter-laboratory differences, varying susceptibilities and metabolic capabilities between species used, and differences in techniques and exposure routes. As a result, the only studies that are used to make relative potency estimates meet the criteria that B[a]P was tested in the same bioassay system as the other PAHs, in the same laboratory, and at the same time. In this section, individual studies that meet these criteria are summarized and evaluated regarding quality, and relative potency estimates are derived for each of the PAHs for which adequate data are available. In some instances, the studies were conducted with several PAHs in addition to those commonly encountered at hazardous waste sites. In these studies, the results for the other PAHs are included for comparison. For each study discussed, a table is included that summarizes all of the results obtained; another table presents the data actually used to derive the relative potency estimates, for ease of verification. The order in which the studies are discussed corresponds to the reverse of the chronological order in which they were performed, with the most recent study presented first. Relative potency estimates are derived by the method described in Section III.

1. Deutsch-Wenzel et al. (1983)

In this study, eight environmentally ubiquitous PAHs were tested for carcinogenicity by direct implantation into the lungs of female Osborne-Mendel rats. Test compounds were prepared in solution with trioctanoin and molten beeswax. Upon injection into the left lobe of the lung, the mixture congealed into a pellet, from which the test compound diffused over time into the surrounding lung tissue. Epidermoid carcinomas and pleomorphic sarcomas were observed and dose-response relationships were

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obtained. The treatment method involved some trauma to the animals. Relative potency estimates would not be affected, however, since this factor was constant for all treatment groups. Tumor induction times were difficult to observe, so survival time and the number of tumor-bearing animals were the responses evaluated. Table IV-1 shows the results for the PAHs tested.

The transition rate parameter R_jS was estimated for each of the carcinogenic PAHs from the data in Table IV-1. The measure of response used for modeling was the number of animals bearing epidermoid carcinomas of the lung at the completion of the study. (Pleiomorphic sarcomas were excluded in the interest of consistency and because their etiology probably differs from that of carcinomas.) The numerical form of the best-fitting model and the goodness of fit are shown in Table IV-2. No tumors were observed in the control rats, so the parameter A was obtained by Bayesian estimation, as discussed in Section III (i.e., $A = -\ln(1-.5/36) = 0.014$). The highest exposure groups were omitted for B[a]P and IND, since the response was significantly less than that predicted by the model. This discrepancy is most likely due to toxicity and reduced survival in those high exposure groups. A more complex analysis could use median survival times as an additional variable; this would most likely reduce the relative potency estimates somewhat and increase precision.

Note that the low-dose linear two-stage model yields potency estimates ranging from slightly more than one to three

TABLE IV-1

CARCINOGENICITY AND DOSE-RESPONSE RELATIONSHIPS OF EIGHT FREQUENTLY OCCURRING ENVIRONMENTAL PAHs IN RAT LUNGS

Compound[a]	Dose (mg)	Number of Animals	Median Survival Time, Weeks (95% Confidence Interval)	Number of Animals Bearing Epidermoid Carcinomas/ Number of Animals Bearing Pleomorphic Sarcomas	Tumor Incidence (%)
B[b]F	0.1	35	110(95-118)	0/1	2.9
B[b]F	0.3	35	113(100-121)	1/2	8.6
B[b]F	1.0	35	112(98-126)	9/4	37.1
B[e]P	0.2	35	117(105-123)	0/0	0.0
B[e]P	1.0	30	111(91-126)	0/1	3.3
B[e]P	5.0	35	104(92-113)	1/0	2.9
B[j]F	0.2	35	110(91-118)	1/0	2.9
B[j]F	1.0	35	117(103-125)	3/0	8.6
B[j]F	5.0	35	89(75-105)	18/0	51.4
B[k]F	0.16	35	114(101-122)	0/0	0.0
B[k]F	0.83	31	95(81-104)	3/0	9.7
B[k]F	4.15	27	98(87-108)	12/0	44.4
IND	0.16	35	116(95-123)	3/1	11.4
IND	0.83	35	109(98-116)	8/0	22.9
IND	4.15	35	92(82-108)	21/0	60.0
ANT	0.16	35	102(91-116)	1/0	2.9
ANT	0.83	35	88(72-103)	19/0	54.3
B[ghi]P	0.16	35	109(101-121)	0/0	0.0
B[ghi]P	0.83	35	114(103-123)	1/0	2.9
B[ghi]P	4.15	34	106(93-117)	4/0	11.8
B[a]P	0.1	35	111(95-120)	4/6	28.6
B[a]P	0.3	35	77(68-99)	21/2	65.7
B[a]P	1.0	35	54(46-64)	33/0	94.3
BW-TC	--	35	104(91-121)	0/0	0.0
Untreated control	--	35	118(104-133)	0/0	0.0

[a] Abbreviations: B[b]F, benzo[b]fluoranthene; B[e]P, benzo[e]pyrene; B[j]F, benzo[j]fluoranthene; B[k]F, benzo[k]fluoranthene; IND, indeno(1,2,3-cd)pyrene; ANT, anthanthrene; B[ghi]P, benzo[ghi]perylene; B[a]P, benzo[a]pyrene; BW-TC, beeswax and trioctanoin (vehicle).

SOURCE: Deutsch-Wenzel et al. (1983).

ABLE IV-2

DATA USED AND ESTIMATES OF RELATIVE POTENCY OBTAINED FROM RAT LUNGS FOLLOWING INTRAPULMONARY INJECTION

Compound[a] Exposure Levels in mg (x)	No. Rats Exposed	Epidermoid Carcinomas		Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)	Deutsch-Wenzel Relative Potency Estimates Using Probit Model
		Observed	Predicted			
B[a]P:						
0.1	35	4	4.79	22.28	1.	1.
0.3	35	21	19.82			
1.0	35	([b])	([b])			
B[b]F:						
0.1	35	0	0.83	3.12	0.140	0.11
0.3	35	1	1.73			
1.0	35	9	7.40			
B[k]F:						
0.16	35	0	0.75	1.48	0.066	0.03
0.83	31	3	2.09			
4.15	27	12	13.79			
IND:						
0.16	35	3	1.66	5.17	0.232	0.08
0.83	35	8	11.41			
4.15	35	([a])	([a])			
B[ghi]P:						
0.16	35	0	0.57	0.486	0.022	0.01
0.83	35	1	0.95			
4.15	34	4	4.07			
Control						
0.00	35	0	--	--	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[b]F, benzo[b]fluoranthene; B[k]F, benzo[k]fluoranthene; IND, indeno(1,2,3-cd)pyrene; B[ghi]P, benzo[ghi]perylene.

[b] Omitted because of lack of fit.

Model: $P(x) = 1-\exp-A(1+SRj)^2$, $A = -\ln(1-.5/36) = 0.014$.

Source: Deutsch-Wenzel et al. (1983).

times greater than, although within the confidence limits of, those obtained by the authors of the study based on the probit model.

2. LaVoie et al. (1982)

The tumor-initiating activities of the benzofluoranthenes were tested on Swiss albino CD-1 female mouse skin and compared with that of B[a]P. Compounds were applied in 10 doses (1 every other day) to the backs of mice and followed 10 days later with applications of the tumor promoter phorbol myristate acetate (TPA) 3 times a week for 20 weeks. Table IV-3 shows the total initiating dose of each of the compounds tested and numbers of tumors and tumor-bearing animals obtained. Tumors were principally squamous cell papillomas, with some keratoacanthomas observed as well. Benzo[b]fluoranthene was approximately ten times as potent as a tumor initiator than benzo[j]fluoranthene, which was only somewhat more potent than benzo[k]fluoranthene. B[a]P was more potent than all three.

Two factors make the data obtained in this experiment difficult to interpret. The application of the promotor TPA adds an additional element of complexity to the evaluation. The possibility of a qualitatively different interaction between TPA and the various PAHs exists. Furthermore, this experimental protocol may allow only the first and not the second mutation that is hypothesized to occur during PAH-induced carcinogenesis and would therefore not fit a two-stage model. These potential problems are compounded by having only a single level of B[a]P

TABLE IV-3

TUMOR INITIATING ACTIVITY OF BENZOFLUORANTHENES

Compound[a]	Total Initiating Dose (μg)	Number of Skin Tumor-Bearing Animals/Number of Animals	Skin Tumors per Animal
B[b]F	100	16/20	7.1
B[b]F	30	12/20	2.3
B[b]F	10	9/20	0.9
B[j]F	1,000	19/20	7.2
B[j]F	100	11/20	1.9
B[j]F	30	6/20	0.6
B[k]F	1,000	15/20	2.8
B[k]F	100	5/20	0.4
B[k]F	30	1/20	0.1
B[a]P	30	17/20	4.9
Acetone (Vehicle)	--	0/20	0

[a] Abbreviations: B[b]F, benzo[b]fluoranthene; B[j]F, benzo[j]fluoranthene; B[k]F, benzo[k]fluoranthene; B[a]P, benzo[a]pyrene.

Source: LaVoie et al. (1982).

available upon which to base the potency estimate. To minimize the latter problem, the data used in the evaluation will be restricted to the single exposure level of each of the PAHs that gives a tumor response closest to the 85% B[a]P tumor response rate. This procedure yields results that are less model-dependent. The relative potencies calculated on the basis of these restricted data are displayed in Table IV-4.

3. Habs et al. (1980)

Six environmentally relevant PAHs were tested in order to determine their dose-response relationships as carcinogens when topically applied to the backs of female NMRI mice throughout their lifetimes. Table IV-5 lists the compounds tested, doses used, frequency of application, and results obtained. Clear-cut dose-response relationships were seen for B[a]P and benzo[b]-fluoranthene. Benzo[j]fluoranthene proved to be weakly carcinogenic, as did cyclopentadieno(c,d)pyrene and coronene. Benzo[k]fluoranthene and indeno(1,2,3-cd)pyrene were inactive as carcinogens in this test system.

The relative transition rate using the low-dose linear two-stage model was calculated for each of the six test agents, as shown in Table IV-6. The estimates obtained for B[j]F, CP, B[k]F, and IND are based upon very weak or perhaps nonexistent dose-response relationships. Even so, the response data observed are consistent with the underlying response model. Estimates obtained using these data would, if erroneous, tend to overestimate the relative transition rates and, as a result, the

TABLE IV-4

DATA USED AND ESTIMATES OF RELATIVE POTENCY OBTAINED FROM MOUSE SKIN INITIATION-PROMOTION EXPERIMENT

Compound[a]	Total Dose (μg)	Number of Skin Tumor-Bearing Animals/ Number Exposed	Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
B[a]P	30	17/20	0.263	1
B[b]F	100	16/20	0.0719	0.273
B[j]F	1,000	19/20	0.0102	0.039
B[k]F	1,000	15/20	0.0066	0.025

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[b]F, benzo[b]fluoranthene; B[j]F, benzo[j]fluoranthene; B[k]F, benzo[k]fluoranthene.

Model: $P(x) = 1\text{-}\exp\text{-}A(1 + SRj)^2$, $A = -\ln[1-.5/21] = 0.014$.

Source: LaVoie et al. (1982).

TABLE IV-5

PAH-INDUCED SKIN TUMOR RATES IN MICE

Compound[a]	Solvent	Frequency of Application	Dose (µg/animal)	Number of Tumor-Bearing Animals/ Total Number of Animals	Tumor Incidence (%)
B[a]P	Acetone	2x/week	1.7	8/34	23.5
			2.8	24/35	68.6
			4.6	22/36	61.1
B[b]F	Acetone	2x/week	3.4	2/38	5.3
			5.6	5/34	14.7
			9.2	20/37	54.1
B[j]F	Acetone	2x/week	3.4	1/38	2.6
			5.6	1/35	2.9
			9.2	2/38	5.3
B[k]F	Acetone	2x/week	3.4	1/39	2.6
			5.6	0/38	0
			9.2	0/38	0
IND	Acetone	2x/week	3.4	1/36	2.8
			5.6	0/37	0
			9.2	0/37	0
CP	Acetone	2x/week	1.7	0/34	0
			6.8	0/35	0
			27.2	3/38	7.9
COR	DMSO	4x/week	5.0	1/39	2.6
			15.0	2/40	5.0
--	Acetone	2x/week	--	0/35	0
--	DMSO	4x/week	--	0/36	0

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[b]F, benzo[b]fluoranthene; B[j]F, benzo[j]fluoranthene; B[k]F, benzo[k]fluoranthene; IND, indeno[1,2,3-cd]pyrene; CP, cyclopentadieno(cd)pyrene; COR, coronene; DMSO, dimethylsulfoxide.

Source: Adapted from Habs et al. (1980).

DATA USED AND ESTIMATES OF RELATIVE POTENCY OBTAINED FROM MICE S PAINTING STUDY

Compound[a] Dose µg/animal (x)	No. Mice Exposed	No. of Tumor-Bearing Animals		Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)	Habs et al. (1980) Relative Potency Estimates Using Probit Model
		Observed	Predicted			
B[a]P:				3.92	1.	1.
1.7	34	8	10.36			
2.8	35	24	20.60			
4.6	--	(b)	(b)			
B[b]F:				0.656	0.167	0.27
3.4	38	2	2.38			
5.6	34	5	4.30			
9.2	--	(b)	(b)			
B[j]F:				0.241	0.061	--
3.4	38	1	0.77			
5.6	35	1	1.18			
9.2	38	2	2.36			
CP:				0.091	0.023	0.05
1.7	34	0	0.28			
6.8	35	0	0.56			
27.2	38	3	2.74			
B[k]F:				0.078	0.020	--
3.4	39	1	0.43			
5.6	38	0	0.59			
9.2	38	0	0.86			
IND:				0.081	0.021	--
3.4	36	1	0.36			
5.6	37	0	0.48			
9.2	37	0	0.69			

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[b]F, benzo[b]fluoranthene; B[j]F, benzo[j]fluoranthene; CP, cyclopentadieno(cd)pyrene; B[k]F, benzo[k]fluoranthene; IND, indeno[1,2,3-cd]pyrene.

[b] Omitted because of lack of fit.

Model: $P(x) = 1 - \exp - A(1+SRj)^2$, $A = -\ln(1-.5/81) = 0.006192$.

Source: Habs et al. (1980).

relative potencies because the actual tumor response may be much lower or non-existent. Note that the relative potency estimates obtained using this model are about a factor of two smaller than those obtained by the author based upon the log-probit model. This difference is primarily due to the elimination of the high-dose information for B[a]P that gave a response that was lower than expected for no directly discernible reason.

4. Pfeiffer (1977)

As part of an experiment designed to evaluate the interactions between carcinogenic and noncarcinogenic PAHS, single doses of B[a]P or dibenz[ah]anthracene dissolved in tricaprylin were injected subcutaneously between the shoulder blades of female NMRI mice. Animals were palpated weekly for tumor development. Table IV-7 shows the incidence of sarcomas obtained and the doses of PAHs used. The control tumor incidence was not reported, although a group of "non-carcinogenic" PAHs was tested and will be included here as a pseudo-control group. Furthermore, the numbers of effective or surviving animals were not reported, so the tumor incidences may not reflect potential toxicity at the higher doses.

The dose-response curves for B[a]P and D[ah]A increase at rates that are less than linear, presumably due either to a reduced rate of absorption or saturation of metabolic activating systems at high doses. This effect is more pronounced for D[ah]A than B[a]P, so that relative potencies for D[ah]A estimated at high doses are lower than those estimated at lower

TABLE IV-7

TUMOR INCIDENCE FOLLOWING SUBCUTANEOUS INJECTION OF BENZO[A]PYRENE OR DIBENZ[AH]ANTHRACENE

Compound[a]	Dose (μg)	Number of Tumor-Bearing Animals Number of Animals	Tumor Incidence (%)
B[a]P	3.12	9/100	9
	6.25	35/100	35
	12.5	51/100	51
	25.0	57/100	57
	50.0	77/100	77
	100.0	83/100	83
D[ah]A	2.35	37/100	37
	4.7	39/100	39
	9.3	44/100	44
	18.7	56/100	56
	37.5	65/100	65
	75.0	69/100	69
Pseudo-control[b]	273.975	6/100	6
	549	8/100	8
	1,098.37	6/100	6
	2,196.75	4/100	4
	4,394	13/100	13
	8,787	5/100	5

[a] B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene.

[b] 10 "noncarcinogenic" PAHs. The "noncarcinogenic" PAH group included chrysene, which is generally considered to be weakly carcinogenic; at the dose levels used, however, chrysene alone cannot account for the tumor response observed.

NOTE: Control animals were included in the experiment; however, whether they were vehicle or untreated controls was not specified, and their tumor incidence was not reported.

Source: Pfeiffer (1977).

doses. The lack of a true control population and the presence of a tumor response from the population receiving a group of hypothesized noncarcinogenic PAHs also make fitting the data to the low-dose linear two-stage model difficult to interpret. As a result, the relative potency was determined by relating results at comparable observed response levels and using the noncarcinogenic PAHs as the pseudo-vehicle control. The data used for these comparisons are shown in Table IV-8. Two estimates of the relative potency were obtained; however, the estimate corresponding to the lower exposure levels (i.e., 2.81) is probably more reasonable to use for low-dose extrapolations.

5. Bingham and Falk (1969)

Graded concentrations of B[a]P or benz[a]anthracene (B[a]A) were applied topically to the backs of C3H/He mice 3 times a week for 50 weeks and local tumors were quantitated. B[a]P was dissolved in decalin, benz[a]anthracene was dissolved in toluene and 50 mg by weight of each solution was administered at each dosing; however, the method of application was not specified. Table IV-9 shows the results of this experiment. No solvent controls were included. From these results, benz[a]anthracene appears to be less potent than B[a]P, although the use of different solvents for each agent introduces a confounding factor that cannot be directly evaluated.

An extremely shallow dose-response relationship for B[a]A was obtained over a two order-of-magnitude dose range. The most likely explanation for this response is that the administered

TABLE IV-8

DATA USED AND ESTIMATES OF RELATIVE POTENCY OBTAINED FROM SUBCUTANEOUS INJECTION STUDY WITH FEMALE MICE

Compound[a]	Dose (µg) (x)	Number Exposed	Number Mice with Tumors	Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
Low dose:					
B[a]P	6.25	100	35	0.2297	1
D[ah]A	2.35	100	37	0.6480	2.82
High dose:					
B[a]P	25.00	100	57	0.0964	1
D[ah]A	18.70	100	56	0.1264	1.31
Pseudo-control	0.00	600	42	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene.

Model: $P(x) = 1-\exp-A(1+SRj)^2$, $A = -\ln[1-42/600] = 0.0726$.

Source: Pfeiffer et al. (1977).

TABLE IV-9

TUMOR INCIDENCE FOLLOWING DERMAL EXPOSURE OF MICE TO BENZO[A]PYRENE AND BENZ[A]ANTHRACENE

Compound[a]	Dose (% Concentration)	Number of Animals Bearing Malignant Tumors/Number of Animals	Tumor Incidence (%)
B[a]P	0.02	5/12	42
	0.002	0/20	0
	0.0002	0/21	0
	0.00002	0/18	0
B[a]A	1.0	5/29	17
	0.2	3/32	9
	0.02	1/18	6
	0.002	0/32	0

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[a]A, benz[a]anthracene.

Source: Bingham and Falk (1969).

dose and the biologically active dose at the target site (reactive form of the agent at the site of action) are not linearly related because of saturation of metabolic activating systems or permeability. In this case, the responses obtained at the lowest exposure levels would be the most directly comparable between agents. The data used to obtain the relative potency estimate for B[a]A are the single levels of agent giving the lowest response for both B[a]P and B[a]A. The data used and the relative potency estimate derived are shown in Table IV-10. Incorporation of the other exposure levels of B[a]A into the relative potency estimate would decrease the estimate for that agent.

6. Van Duuren et al. (1966)

As part of an experiment to evaluate the tumorigenic potential of tobacco leaf and smoke constituents, several PAHs were tested for tumor initiating activity. The compounds were applied as single doses in 0.1 ml of solvent to the backs of female ICR/Ha Swiss mice. This treatment was followed by applications of 25 µg croton resin in acetone as a promoter three times a week for 63 weeks (or until death). The compounds tested and the experimental results obtained are presented in Table IV-11. B[a]P is clearly more potent than any of the other PAHs tested.

The data used to obtain potency estimates for CH and B[a]F are shown in Table IV-12. The response employed for the estimates was number of malignant tumor-bearing animals by the

TABLE IV-10

DATA USED AND ESTIMATE OF RELATIVE POTENCY OBTAINED FOR BENZ(A)ANTHRACENE FROM MOUSE SKIN PAINTING STUDY

Compound[a] Dose (% concentration (x)	No. Mice Exposed (Survived 50 weeks)	No. Mice with Malignant Tumor	Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
B[a]P:				
.02	12	5	187.0	1.0
B[a]A:				
.02	18	1	27.16	0.145[b]
.20	32	3	5.13	0.027
1.0	29	5	1.81	0.0097
Control[c]:				
0	20	0	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[a]A, benz[a]anthracene.

[b] Value used for relative potency estimate.

[c] Assumed value.

Model: $P(x) = 1-\exp-A(1 + SRj)^2$, $A = -\ln(1-0.5/21) = 0.024$.

Source: Bingham and Falk (1969).

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TABLE IV-11

PAH-INDUCED SKIN TU[illegible]R INITIATION IN MICE USING CROTON RESIN AS A PROMOTER

			Papillomas		Carcinomas	
Compound[a]	Dose	Solvent	Number of Tumor-Bearing Animals/ Number of Animals	Tumor Incidence (%)	Number of Tumor-Bearing Animals/ Number of Animals	Tumor Incidence (%)
B[a]P	150 µg	0.1 ml acetone	20/20	100	6/20	30
CH	1 mg	0.4 ml acetone	16/20	80	2/20	10
B[b]F	1 mg	0.1 ml acetone	18/20	90	5/20	25
B[mno]F	1 mg	0.1 ml acetone	4/20	20	0/20	0
Control (promotion with acetone only)	--	--	5/20	25	1/20	5
Control (untreated)	--	--	0/100	0	0/20	0

[a] Abbreviations: B[a]P, benzo[a]pyrene; CH, chrysene; B[b]F, benzo[b]fluoranthene; B[mno]F, benzo[mno]fluoranthene.

Source: Van Duuren et al. (1966).

TABLE IV-12

DATA USED AND ESTIMATES OF RELATIVE POTENCY OBTAINED FROM MOUSE SKIN INITIATION PROMOTION EXPERIMENT

Compound[a]	Dose[b] (mg)	No. Exposed	No. with Cancer by 63rd Week	Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
B[a]P	0.15	20	6	10.91	1.
CH	1.0	20	2	0.433	0.040
B[b]F	1.0	20	5	1.368	0.125
Promotor control	0	20	1	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; CH, chrysene; B[b]F, benzo[b]fluoranthene.

[b] PAH administration was followed by 25 μg croton resin applied three times weekly for 63 weeks.

Model: $P(x) = 1-\exp-A(1+SRj)^2$, $A = -\ln(1-1/20) = 0.0513$.

Source: Van Duuren et al. (1966).

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63rd week of the test. The necessity of using a promoter to obtain a carcinogenic response in the test system is a factor that would tend to reduce the credibility of the potency estimates if no other information was available, although since internal comparisons are available for the determination of consistency, using the initation-promotion system can supply valuable additional information. Use of the two-stage model to evaluate an initiation/promotion experiment is questionable, however. The potency estimate for CH in this system is highly variable, since the response level is not clearly different from the croton resin control.

7. Hoffmann and Wynder (1966)

Two different types of experiment were performed:

(1) "Complete" carcinogenesis experiments, in which PAHs were topically administered for 1 year and

(2) Initiation-promotion experiments, in which PAHs were administered for only 3 weeks, followed by application of a tumor-promoting agent for the duration of the experiment.

For the "complete" carcinogenicity evaluations, female Ha/ICR/mil Swiss albino mice received three weekly topical applications of B[a]P, benzo[ghi]perylene, or indeno(1,2,3-cd)pyrene for one year. The number of papillomas observed at each dose for each group is shown in Table IV-13. B[a]P and benzo[ghi]perylene were dissolved in dioxane, and indeno(1,2,3-cd)pyrene was dissolved in acetone. No tumors were observed in the dioxane vehicle controls; no acetone control was included. The results

TABLE IV-13

CARCINOGENIC ACTIVITY OF SEVERAL PAHs ON MOUSE SKIN

Compound[a]	Dose (% Concentration)	Number of Tumor-Bearing Animals/ Number of Animals	Tumor Incidence (%)
B[a]P	0.05	17/20	85
	0.1	19/20	95
IND	0.01	0/20	0
	0.05	0/20	0
	0.1	6/20	30
	0.5	7/20	35
B[ghi]P	0.05	1/20	5
	0.1	0/20	0

[a] Abbreviations: B[a]P, benzo[a]pyrene; IND, indeno(1,2,3-cd)pyrene; B[ghi]P, benzo[ghi]perylene.

Source: Hoffmann and Wynder (1966).

show that B[a]P is much more potent as a carcinogen than either of the other PAHs tested in this model.

For the initiation-promotion experiments, ten doses of the same PAHs tested above were applied in dioxane 2 days apart to the backs of mice for a total dose of 0.25 mg per mouse and followed by 2.5% croton oil in acetone. The frequency and duration of croton oil administration were unclear. The results of these experiments are found in Table IV-14. Again, B[a]P was shown to be much more potent than either of the other PAHs tested.

For each of the compounds used in the first experiment, the lowest dose that gave a positive response was employed in the relative potency estimation. Again, since B[a]P gave a much higher response than the other PAHs and the slope appears to be very low, the potency estimate, if in error, is most likely overestimated. These estimates are shown in Table IV-15.

The initiation-promotion experiment yielded results that would tend to overestimate risk for two reasons. First, the responses for IND and B[ghi]P may be spurious, since they are not statistically elevated over the control response; second, the response for B[a]P is in a high range in which a lower percent of the applied dose may be converted to a biologically active target dose, as compared with lower applied dose levels. The relative potency estimates obtained from this experiment are displayed in Table IV-16, although they are probably less reliable than those obtained in the "complete" carcinogenses

TABLE IV-14

INITIATION-PROMOTION ACTIVITY OF SEVERAL PAHs ON MOUSE SKIN

Compound[a]	Total Dose[b] (mg)	Number of Tumor-Bearing Animals/ Number of Animals	Tumor Incidence (%)
B[a]P	0.25	24/30	80
IND	0.25	5/30	17
B[ghi]P	0.25	2/27	7
Control[c]	0	2/30	7

[a] Abbreviations: B[a]P, benzo[a]pyrene; IND, indeno(1,2,3-cd)pyrene; B[ghi]P, benzo[ghi]perylene.

[b] Administration of PAHs was followed by 2.5% croton oil as a promoter.

[c] Croton oil was administered in the absence of PAH treatment.

Source: Hoffmann and Wynder (1966).

TABLE IV-15

DATA USED AND ESTIMATES OF RELATIVE POTENCY OBTAINED FROM MOUSE SKIN COMPLETE CARCINOGENESIS EXPERIMENT

Compound[a]	Dose (% Concentration) (x)	No. of Animals Exposed	No. of Tumor-Bearing Animals	Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
B[a]P	0.05	20	17	157.8	1.0
IND	0.10	20	6	28.55	0.181
B[ghi]P[b]	0.05	20	1	9.238	0.0585
Control	0	20	0	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; IND, indeno(1,2,3-cd)pyrene; B[ghi]P, benzo[ghi]perylene.

[b] Response was not dose related.

Model: $P(x) = 1-\exp-A(1+SRj)^2$, $A = -\ln(1-.5/21) = .0241$.

Source: Hoffman and Wynder (1966).

TABLE IV-16

DATA USED AND ESTIMATES OF RELATIVE POTENCY OBTAINED FROM MOUSE SKIN INITIATION-PROMOTION EXPERIMENT

Compound[a]	Dose[b] (ng)	No. of Animals Exposed	No. of Tumor-Bearing Animals	Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
B[a]P	0.25	30	24	15.32	1.0
IND[c]	0.25	30	5	2.502	0.163
B[ghi]P[c]	0.25	27	2	0.224	0.0147
Control	0	30	2	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; IND, indeno(1,2,3-cd)pyrene; B[ghi]P, benzo[ghi]perylene.

[b] The PAH dose was followed by 2.5% croton oil as a promoter.

[c] Response was not statistically elevated over control.

Model: $P(x) = 1\text{-exp-}A(1+SRj)^2$, $A = -\ln(1-2/30) = 0.0690$.

Source: Hoffman and Wynder (1966).

experiment because of the inconsistency of the initiation-promotion protocol and the two-stage model.

8. Wynder and Hoffmann (1959)

As part of a study of the carcinogenicity of tobacco and its constituents, several PAHs were tested as complete carcinogens on the skin of mice. Female Swiss mice received various concentrations of the test substances dissolved in acetone three times a week applied to their backs with a brush throughout their lifetimes. Table IV-17 shows the compounds tested, concentrations used, and the results of these experiments. No solvent control group was reported; however, since no papillomas or carcinomas were obtained for several of the PAHs tested, a solvent control group would most likely have been negative as well. The results indicate that B[a]P and dibenz[ah]anthracene are somewhat similar in potency in this model and chrysene is much less potent.

In this experiment, the survival rate was much less for B[a]P-treated animals than for those receiving the other PAHs. To adjust for the difference in survival rates, the relative potency estimates will be based on comparisons of tumor rates observed at 14 months using number of animals bearing carcinomas as the response. The data used and the relative potency estimates generated for D[ah]A and CH are shown in Table IV-18. Estimates obtained from this experiment are considered to be of high quality because of two factors: First, the response spans the full range from zero to almost 100% for both

TABLE IV-17

CARCINOGENIC ACTIVITY OF PAHs ON MOUSE SKIN

Compound[a]	Dose (% Concentration)	Incidence of Papillomas (%)	Incidence of Carcinomas (%)
B[a]P	0.001	43	3
	0.005	73	63
	0.01	95	95
D[ah]A	0.001	30	30
	0.01	95	90
	0.1	90	75
CH	1.0	45	40
AN	10.0	0	0
PY	10.0	0	0
FL	0.1	0	0

[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene; CH, chrysene; AN, anthracene; PY, pyrene; FL, fluoranthene.

Source: Wynder and Hoffmann (1959).

TABLE IV-18

DATA USED AND RELATIVE POTENCIES OBTAINED FROM MOUSE SKIN CARCINOGENESIS EXPERIMENT

Compound[a] Dose (% concentration)(x)	No. Animals Exposed	No. Animals with Carcinomas by 14 Months		Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
		Observed	Predicted		
B[a]P:				822.0	1.0
.001	29	0	2.23		
.005	30	19	14.01		
.01	20	19	17.42		
D[ah]A:				912.0	1.11
.001	20	2	1.69		
.01	20	18	18.31		
.1	(b)	(b)	(b)		
CH:					
1.	20	8	8.00	3.60	0.0044
Control:					
0	20	0	0.48	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene; CH, chrysene.

[b] Omitted because of lack of fit.

Model: $P(x) = 1-\exp-A(1+SRj)^2$, $A = -\ln(1-.5/21) = 0.0241$.

Source: Wynder and Hoffman (1959).

B[a]P and D[ah]A; second, the model is consistent with the data for the full range of B[a]P exposures.

9. Bryan and Shimkin (1943)

A study was conducted to establish dose-response curves for several PAHs following subcutaneous injection into the right axilla of male C3H mice. The solvent used was tricaprylin, although no solvent control group was reported. Animals were palpated frequently following a single injection of the test compound in order to detect tumors; 99% of those detected proved to be spindle-cell sarcomas (the remaining 1% were carcinomas or mixed). Table IV-19 shows the results obtained for B[a]P and dibenz[ah]anthracene. Dibenz[ah]anthracene appears to be somewhat more potent than B[a]P in this system.

Although this classic experiment with 12 exposure levels per compound gave a full range of response for each compound, several factors make it difficult to interpret. An exposure level of 0.0039 mg appears to have been included for both B[a]P and D[ah]A; however, no results were reported for this dose, and no explanation for the omission was included. Vehicle control response levels were also not reported. This problem is exacerbated by the abnormality of a positive response at the lowest exposure level for B[a]P (0.00195 mg) in the absence of a response at exposure levels 4, 8, or 16 times higher. For the analysis conducted here, the data will be restricted to the monotonically increasing portion of the curve, which is similar to the approach taken by the authors in their fitting of the

TABLE IV-19

TUMOR INCIDENCE OBSERVED IN MICE FOLLOWING SUBCUTANEOUS INJECTION OF BENZO[A]PYRENE OR DIBENZ[AH]ANTHRACENE

Compound[a]	Dose (mg)	Number of Tumor-Bearing Animals/ Number of Animals	Tumor Incidence (%)
D[ah]A	8.0[b]	16/21	76
	4.0	17/20	85
	2.0	19/19	100
	1.0	22/22	100
	0.5	20/21	95
	0.25	19/21	90
	0.125	21/23	91
	0.062	20/20	100
	0.031	16/21	76
	0.0156	6/19	32
	0.0078	6/40	15
	0.00195	2/79	3
B[a]P	8.0	20/21	95
	4.0	16/19	84
	2.0	19/19	100
	1.0	18/20	90
	0.5	19/19	100
	0.25	14/21	67
	0.125	15/19	79
	0.062	4/20	20
	0.031	0/16	0
	0.0156	0/19	0
	0.0078	0/40	0
	0.00195	2/81	2

[a] D[ah]A, dibenz[ah]anthracene; B[a]P, benzo[a]pyrene.

[b] Volume of solvent was 0.5 ml for the 8.0 mg dose of D[ah]A and 0.25 ml for all other doses.

Source: Bryan and Shimkin (1943).

probit model. The only difference is that the authors of the study included the 0.125 mg level for D[ah]A in their analysis, ich would have the effect of causing a slight decrease in the relative potency estimates and yielding a worse fit. In addition, for the purpose of the present analysis, the assumption will be made that the lowest dose of B[a]P (0.00195 mg) actually represents a control level. This assumption is consistent with the observed response data. The dose-response data used in the potency determination are shown in Table IV-20. Under the conditions of this experiment, the potency of D[ah]A is estimated to be 4.5 times that of B[a]P.

B. SURROGATE APPROACHES: DNA ADDUCT FORMATION

Interaction between reactive PAH metabolites and DNA has been established as a necessary (albeit not sufficient) event for PAH-induced tumorigenesis (Gelboin 1980, Weinstein et al. 1978). The formation of B[a]P diol-epoxide DNA adducts has been shown to be linearly related to administered dose for several organs in rats and mice (Pereira et al. 1979, Adrienssens et al. 1983), and related to a quadratic increase in tumor formation (Lee and O'Neill 1971). It is reasonable to suggest that in the absence of quantitative tumor dose-response information for carcinogenic PAHs that can be readily compared to B[a]P, the formation of DNA adducts could be used as a surrogate basis for establishing relative potency estimates.

It is recognized that the DNA adduct level in a tissue is not directly related to carcinogenic response. Other factors

TABLE IV-20

DATA USED TO OBTAIN A RELATIVE POTENCY ESTIMATE FOR DIBENZ[AH]ANTHRACENE FROM A SUBCUTANEOUS INJECTION EXPERIMENT

Compound[a] Dose (mg)	No. of Mice Exposed	Tumors		Estimated Transition Rate Parameter (SRj)	Estimated Relative Potency (Rj)
		Observed	Expected		
B[a]P:				42.8	1.0
0.125	19	15	12.15		
0.062	20	4	5.73		
0.031	16	0	2.05		
D[ah]A:				192.4	4.50
0.062	20	20	19.71		
0.031	21	16	14.84		
0.0156	19	6	6.33		
0.0078	40	6	5.85		
Pseudo-control[b]:					
0.00195	160	4	4.0	--	--

[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene.

[b] Combined response at a dose level of 0.00195 mg of B[a]P or D[ah]A.

Model: $P(x) = 1-\exp-A(1+SRj)^2$, $A = -\ln(1-4/160) = 0.0253$.

Source: Bryan and Shimkin (1943).

such as the DNA repair and cell turnover rates in the target tissue can have a greater effect than the actual number of adducts on the ultimate probability of tumor formation. However, under the assumptions of the current model of PAH tumor induction, the transition rate is linearly related to the DNA adduct formation rate. As a result, the relative potency measured as the ratio of DNA adducts per unit administered dose for two PAHs is assumed to be independent of tissue type, route of exposure, and test system as long as the ratios of the levels of distribution, metabolism, and elimination for B[a]P and the PAH in question remain unchanged. Furthermore, DNA adduct formation may reflect cancer risk from PAHs at low, environmental levels of exposure more accurately than dose-response curves based on tumor rates produced at high levels of exposure in the laboratory, when additional biological mechanisms may play a role in enhancing or suppressing tumor rates.

This section describes the studies in which the levels of DNA adduct formation were determined for several PAHs, including B[a]P. The studies are described in reverse chronological order, and relative potencies are determined for the PAHs tested.

1. Reddy et al. (1984)

A total of 28 compounds were tested for ability to bind to DNA using a very sensitive technique (detection limit~~y~~ of 1 aromatic DNA adduct in 10^8 normal adducts) for the detection of DNA adducts employing ^{32}P-postlabelling following X administration to rats and mice. The PAHs tested were applied to the dorsal skin of female BALB/c mice as four doses of 1.2 umole each in acetone at 0, 6, 30, and 54 hours. Animals were sacrificed 24 hours after the last treatment. DNA was isolated from treated skin, digested, enzymatically labelled with ^{32}P (using T4 polynucleotide kinase and $[\gamma-^{32}P]ATP$), resolved using thin-layer chromatography and detected using autoradiography and scintillation counting. Relative adduct labelling was determined by comparing the radioactivity of the adducts to that of normal nucleotides. Absolute levels of adduct formation were not given, and results were expressed as the ranges depicted in Table IV-21; as a result, point estimates of relative potency are not obtainable. The only quantitative conclusion that can be drawn from this study is that B[a]P is the same potency or up to three orders of magnitude more potent than the other PAHs tested. The authors noted, however, that there was a good correlation between adduct level and carcinogenic potency on mouse skin, and that the noncarcinogens failed to form detectable adducts.

2. Phillips et al. (1979)

The levels of binding of seven radioactively-labelled PAHs to mouse skin DNA following topical application were determined. One umole of each PAH in acetone (see Table IV-22 for compounds tested) was applied to the backs of male C57BL mice and the animals were sacrificed at the time of maximal DNA binding (from 19 to 72 hours following administration, depending

TABLE IV-21

RELATIVE DNA BINDING OF PAHs TO MOUSE SKIN

Compound[a]	Relative Level of Binding[b]
B[a]P	+++
B[a]A	++
D[ah]A	++
B[ghi]P	++
CH	++
AN	ND
PY	ND

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[a]A, benz[a]anthracene; D[ah]A, dibenz[ah]anthracene; B[ghi]P, benzo[ghi]perylene; CH, chrysene; AN, anthracene; PY, pyrene.

[b] Relative binding levels: +++, 1 adduct in 10^4 - $5x10^5$ nucleotides; ++, 1 adduct in $5x10^5$ - 10^7 nucleotides.

ND = not detected; detection level = 1 adduct in 10^8 nucleotides.

Source: Reddy et al. (1984).

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TABLE IV-22

EXTENT OF PAH BINDING TO DNA

Compound[a]	Extent of Reaction with DNA (pmole/mg DNA)	Estimated Relative Potency $\frac{\text{pmoles DNA-PAH/mg DNA}}{\text{pmoles DNA-B[a]P/mg DNA}}$ (R_j)
B[a]A	2	0.07
D[ac]A	10	0.37
D[ah]A	15	0.56
7-MBA	25	0.93
3-MC	25	0.93
B[a]P	27	1.0
7,12-DMBA	43	1.6

[a] Abbreviations: B[a]A, benz[a]anthracene; D[ac]A, dibenz[ac]anthracene; D[ah]A, dibenz[ah]anthracene; 7-MBA, 7-methylbenz[a]anthracene; 3-MC, 3-methylcholanthrene; B[a]P, benzo[a]pyrene; 7,12-DMBA, 7,12-dimethylbenz[a]anthracene.

Source: Phillips et al. (1979).

on the compound, which for the purpose of the model is assumed to be proportional to steady-state binding levels under conditions of continuous exposure). DNA was isolated, hydrolyzed enzymatically and chromatographed; levels of PAH binding were determined from the amount of radioactivity eluting with the fractions containing DNA-bound PAHs. The extent of DNA binding for the PAHs tested is shown in Table IV-22.

7,12-Dimethylbenz[a]anthracene, considered to be the most carcinogenic PAH, was found to bind to DNA to the greatest extent; the weakest carcinogens, benz[a]anthracene and dibenz[a,c]anthracene, were bound the least. The extent of binding of the intermediate compounds also appeared to correlate well with what are thought to be their carcinogenic potencies. B[a]P is approximately two orders of magnitude and twice as potent, respectively, as the other PAHs tested, benz[a]anthracene and dibenz[ah]anthracene.

3. Grover and Sims (1968)

The extent to which PAHs react with DNA was determined using an *in vitro* system comprised of a microsomal enzyme activation system, salmon sperm DNA, and 0.5 µg of radioactively labeled PAHs. Following incubation for one hour, the amount of PAH bound to DNA was determined using scintillation counting. The PAHs tested and results obtained are shown in Table IV-23. B[a]P was again shown to be the most active PAH; it was at least twice as potent as the other indicator PAHs.

TABLE IV-23

BINDING OF PAHs TO DNA IN VITRO

Compound[a]	Extent of Reaction with DNA (umoles/mole DNA)	Estimated Relative Potency $\frac{\text{umoles DNA-PAH/mole DNA}}{\text{umoles DNA-B[a]P/mole DNA}}$ (R_j)
B[a]P	1.41	1.0
D[ah]A	0.44	0.31
B[a]A	0.70	0.50
PY	0.31	0.22
PH	0.05	0.04

[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene; B[a]A, benz[a]anthracene; PY, pyrene; PH, phenanthrene.

Source: Grover and Sims (1968).

Establishing a credible biological basis for use of an *in vitro* system on which to base relative potency estimates is difficult, since many of the factors affecting adduct formation *in vivo*, such as DNA repair, are absent; however, the information derived from this system may be used in support of other data.

C. SELECTION OF RELATIVE POTENCY ESTIMATES

In the previous sections, relative potency estimates were derived for carcinogenic PAHs. These estimates were based on the results obtained in ten separate studies using five different experimental systems. The potency estimates and the systems from which they were derived are summarized in Table IV-24. Note that up to four independent estimates of relative potency were obtained for the PAHs.

A composite estimate of the relative potency is required for each PAH. Major qualitative differences exist between the studies from which the estimates are derived concerning their precision and applicability to continuous human ingestion or inhalation exposure. As a result, no attempt will be made to obtain a numerical estimate based upon a weighted average of the studies. Instead, the most relevant study will be selected based upon the following objective criteria and any particular unique condition that is noted. The selection is provisional; a more appropriate method of selection might entail a consensus of opinion from a panel of recognized experts employing a formal system of weighting such as that suggested by DuMouchel and Harris (1983).

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TABLE IV-24
SUMMARY OF RELATIVE POTENCY ESTIMATES FOR INDICATOR PAHs

	TEST SYSTEM				
Compound	Mouse Skin Carcinogenesis	Subcutaneous Injection into Mice	Intrapulmonary Administration to Rats[g]	Initiation-Promotion on Mouse Skin	DNA Adduct Formation[j]
Benzo[a]pyrene	1.0	1.0	1.0	1.0	1.0
Benz[a]anthracene	0.145[a]				0.07
Benzo[b]fluoranthene	0.167[b]		0.140	0.273[h],0.125[i]	
Benzo[k]fluoranthene	0.020[b]		0.066	0.025[h]	
Benzo[ghi]perylene	0.0585[c]		0.022	0.0147[c]	
Chrysene	0.0044[d]			0.040[i]	
Dibenz[ah]anthracene	1.11[d]	2.82[e],4.50[f]			0.56
Indeno[1,2,3-cd]pyrene	0.021[b],0.181[c]		0.232	0.163[c]	

References:
- (a) Bingham and Falk 1969
- (b) Habs et al. 1980
- (c) Hoffmann and Wynder 1966
- (d) Wynder and Hoffmann 1959
- (e) Pfeiffer 1977
- (f) Bryan and Shimkin 1943
- (g) Deutsch-Wenzel et al. 1983
- (h) LaVoie et al. 1980
- (i) Van Duuren et al. 1966
- (j) Phillips et al. 1979

Some of the criteria considered for making the selection are:

(1) The relevance of the route of exposure employed in the bioassay to anticipated human exposure.

(2) The duration of exposure (longer exposures are preferred).

(3) The sample sizes used.

(4) The inclusion of a vehicle control group.

(5) A dose-response relationship obtained that is consistent with the underlying theoretical model, which is linear-quadratic in form.

(6) The extent to which the observed responses cover the entire response range.

(7) The absence of additional complicating variables such as promoters.

The extent to which each of the studies fulfills these criteria, in a relative sense, is indicated in Table IV-25. The scale used is as follows: +++, above average; ++, average; +, below average.

Based on this information and some factors specific to individual studies, the relative potency estimates that are considered to have the most reliable basis were selected and are shown in Table IV-26.

The basis for each of the selections in Table IV-26 is as follows:

Benz[a]anthracene. Only one study was performed from which a relative potency could be derived.

Benzo[b]fluoranthene and Benzo[k]fluoranthene. Both the mouse skin carcinogenesis (Habs et al. 1980) and intrapulmonary administration (Deutsch-Wenzel et al. 1983) studies were found

TABLE IV-25
EXTENT TO WHICH STUDIES USED TO DERIVE RELATIVE POTENCY ESTIMATES FULFILL APPLICABILITY CRITERIA

CRITERIA[a]

Reference	Test System	Route of Exposure	Continuous Exposure	Sample Size	Quality of Controls Used	Absence of Complicating Factors	Full Range of Responses	Fits Dose-Response Model for 2 or More Doses
Deutsch-Wenzel et al. 1983	Intrapulmonary administration	+++	+++	++	+++	++	+++	+++
LaVoie et al. 1980	Initiation-promotion on skin	++	+	+	++	+	+	+
Habs et al. 1980	Skin carcinogenesis	++	+++	++	+++	++	++	+++
Pfeiffer 1977	Subcutaneous injection	+	+	+++	+	++	+++	+
Bingham and Falk 1969	Skin carcinogenesis	++	+++	+	+	++	+	+
Van Duuren et al. 1966	Initiation-promotion on skin	+	+	+	+++	+	+	+
Hoffman and Wynder 1966	Skin carcinogenesis or	++	+++	+	+	++	+	+
	Initiation-promotion on skin	++	+	++	++	+	+	+
Wynder and Hoffman 1959	Skin carcinogenesis	++	+++	+	+	++	+++	+++
Bryan and Shimkin 1943	Subcutaneous Injection	+	+	++	+	++	+++	+++

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[a] Criteria are discussed more fully in the text.

TABLE IV-26
SUMMARY OF RELATIVE POTENCY ESTIMATES DERIVED FOR PAHs

Benzo[a]pyrene	1.0
Benz[a]anthracene	0.145[b]
Benzo[b]fluoranthene	0.140[a]
Benzo[k]fluoranthene	0.066[a]
Benzo[ghi]perylene	0.022[a]
Chrysene	0.0044[c]
Dibenz[ah]anthracene	1.11[c]
Indeno[1,2,3-cd]pyrene	0.232[a]

[a] Deutsch-Wenzel et al. (1983).

[b] Bingham and Falk (1969).

[c] Wynder and Hoffmann (1959).

to be of comparable quality; the estimates based on the latter study were chosen because of the relevance of this route of exposure to that of the B[a]P study on which the dose-response model is based. This choice results in an approximately 15% lower estimate for B[b]F, but in a 3-fold greater estimate for B[k]F.

Benzo[ghi]perylene. IARC (1983) considers this compound to be a noncarcinogen. However, evidence from several studies indicates that it has weak carcinogenic potential. Therefore, for the purpose of conservatism, B[ghi]P will be considered to be carcinogenic and a relative potency estimate derived. The intrapulmonary administration study (Deutsch-Wenzel et al. 1983) proved to fulfill many more of the adequacy criteria than did the skin carcinogenesis study (Hoffman and Wynder 1966), therefore, the former was used as the basis for estimation.

Chrysene. The skin carcinogenesis study (Wynder and Hoffman 1959) was considered to be more appropriate than the initiation-promotion study (Van Duuren et al. 1966) because of the additional variables introduced in the initiation-promotion study. Furthermore, the tumor response observed in the latter study was not statistically significant.

Dibenz[ah]anthracene. On the basis of the extent to which each of the studies of this compound fulfill the criteria for study adequacy, the estimate derived from the skin carcinogenesis study (Wynder and Hoffman 1959) will be used. The relative potency estimate based on DNA adduct formation supports the choice of the lower estimate. Estimates of two to four times higher were obtained from studies that employed subcutaneous injection as a method of administration; however this system is less relevant to human exposure.

Indeno[1,2,3-cd]pyrene. As was true for the benzofluoranthenes, both the Habs et al. (1980) skin carcinogenesis study and the Deutsch-Wenzel et al. (1983) intrapulmonary administration study fulfill most of the adequacy criteria. The estimate based on the latter study was chosen because of its relevance to the route of exposure used in the study of B[a]P from which the dose-response model was derived. Four independent potency estimates were made and the largest chosen. Consistency between estimates was apparent; the three largest estimates varied by no more than a factor of one-third.

V. ESTIMATION OF CANCER RISK DUE TO MIXTURES OF POLYCYCLIC AROMATIC HYDROCARBONS USING ALTERNATIVE METHODS

A number of mathematical models are available that can be used to describe cancer dose-response relationships for PAHs instead of the biologically-based two-stage model described in Section III. These models have several limitations; however, it is useful to see how relative potencies can change depending on the model used and the assumptions made. For the purpose of numerical comparison, this section applies two alternative models (one-hit and multistage) to the tumor data available for PAHs and compares the results to the relative potencies derived using the approach described in this report.

1. One-Hit Model

The one-hit model assumes that the dose-response relationship between PAH exposure and tumor response is one-stage (instead of two-stage or multistage) in form. Under this assumption, only one change is required to turn a normal cell into a tumor cell and the probability of a tumor response may be expressed as

$$P(x) = 1 - \exp -(B_o+B_1x), \qquad \text{(V-1)}$$

where x is the administered dose, B_o is the background tumor rate, and B_1 is the low-dose linear term (i.e., slope of the

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line described by the model at low doses). The one-hit model is a special case of the Armitage-Doll and Guess-Crump models in which time is fixed and only one stage is assumed to be affected.

Given a single bioassay in which a separate tumor dose response relationship is obtained for m different PAHs including B[a]P, the low dose linear term for each PAH (i.e., B_{1i} where $i=1,2,\ldots,m$) can be estimated. The ratio of the linear terms for a specified PAH to the linear term for B[a]P is an estimate of the relative potency of that PAH compared to B[a]P.

The one-hit approach has several advantages. Only one data point is required to obtain a relative potency estimate. The estimates are very stable (i.e., they do not change much as a result of small alterations in the tumor data). The estimates are dose-independent, so they may be used in all dose ranges. The main disadvantages are that many of the PAH dose-response data observed are nonlinear so that systematic biases are introduced by assuming linearity. Also, in the simple form presented in equation V-1, the model can be applied only to studies in which the dose x and time t are constant.

2. Linearized Multistage Model

Point estimates instead of statistical upperbounds for multistage model parameters can be obtained and used to predict tumor rates at various PAH exposure levels. One measure of potency of a particular PAH is the reciprocal of its exposure level that results in a specified tumor response rate. For example, the reciprocal of the exposure level that induced a 10% tumor response based on point estimates from the multistage model is used by EPA as a measure of potency for reportable quantities of potential carcinogens (see Cogliano 1986). The ratio of two potencies so calculated might be used to obtain a relative potency estimate. Relative potencies calculated in this manner may vary at different response levels and as a result, are unreliable because they are highly dependent on the tumor response level chosen. For response levels that are much smaller than those in the observable range (i.e., increases in tumor rates expected at environmental levels of exposure), a very minor shift in the tumor response data in an experiment can be shown to lead to orders of magnitude changes in the relative potency estimates at environmental levels using this approach. As a result, this approach has little to recommend it for assessing low dose potencies over a wide range of potential environmental exposure levels.

Using the 95% upper bound linear term obtained from the multistage model to derive relative potencies instead of point estimates has a superficial level of appeal. This is the measure of potency most often used by EPA in various regulatory settings (see EPA 1986c). The ratios of upper bound estimates are not good measures of relative potency, however, since they are sometimes more dependent upon sample size considerations than on tumor response levels. Since unit risks (i.e., 95% upper bound linear terms) are often used to represent the potencies of carcinogens at environmental levels of exposure, using them to estimate relative potencies is an informative exercise nonetheless.

3. Application of Models

Using the tumor data from Sections III and IV, the parameters in the linearized multistage and one-hit models were estimated and a Chi-square goodness-of-fit statistic obtained. The parameter estimates and X^2 values are shown in Appendix A. In cases where the models did not fit the data, as measured by the approximate X^2 goodness-of-fit test, the tumor rate obtained at the highest dose was omitted and the models were fitted to the reduced data set. Comparisons of the observed and predicted numbers of tumors for each agent in each experiment are shown in Appendix B.

Comparison of the results in Appendices A and B with the corresponding results for the two-stage model presented in Sections III and IV reveals a number of similarities and differences. The one-hit model often yields a poor fit to the data, reflecting the fact that dose-response relationships showed upward curvature in most studies. The multistage model provided adequate fits to the data in all but four cases. In all four of these cases, the two-stage model also fitted poorly to the full data sets and calculations of relative potency were based on selected subsets of the data (Tables IV-4, IV-8, IV-14, and IV-20); in at least of two of these cases, the multistage model gave adequate fits to similar subsets. In a number of cases, the multistage model yielded a linear-quadratic dose-response relationship similar to that assumed in the two-stage model. In other cases, however, the multistage model included only a linear term or the last term in the nth degree polynomial. In several cases, the multistage model yielded slightly better fits to the data than the two-stage model (e.g., Tables B-1, B-3, B-7, and B-9). However, these "improvements" in fit resulted from the larger numbers of parameters in the multistage model with resulting variability in the number of terms (i.e., in the shape of the dose-response curve). With only two exceptions, the two-stage model yielded reasonable fits to the data after elimination of one or more high-dose terms. The exceptions are D[ah]A in Table IV-7 and both D[ah]A and B[a]P, shown in Table IV-19; in these cases a multistage model could not fit the data either. Thus, the available dose-response data are not sufficient to determine which of these two models provides the better description of the data. The two-stage model is preferred for parameter estimation, for two reasons: (a) it has the same form in all cases, whereas the multistage model invokes different numbers of stages to fit different data sets; and (b) it is based on a specific model of the two affected transitions, whereas the multistage model leaves both the nature and the number of the affected transitions unspecified.

Two parameters were used to obtain relative potency estimates: the maximum likelihood estimate of the linear term in the one-hit model and the 95% upper bound linear term in the multistage model, which are also shown in Appendix A. The ratio of each of these parameters to that derived for B[a]P yields the relative potency estimates for the one-hit and multistage models, respectively.

Tables V-1 and V-2 summarize the relative potency estimates derived for each PAH from the bioassay data in Sections III and IV using the one-hit and multistage models. To evaluate these estimates and their consistency as compared to those obtained using the two-stage model, the following analyses were conducted.

For each PAH with three or more estimates of relative potency, the variance of the $\log_{10}$ relative potency was calculated in order to evaluate the consistency of the estimates. The log transformation was used because the relative error is of primary importance, not the absolute variability. The results of this analysis are displayed in Table V-3. The only statistically significant difference (using a standard two sided "F" test at the $p = 0.05$ level) in the estimates is for the comparison of the consistency of the two-stage model versus the one-hit model for the relative potencies of B[b]F. The statistical power of the "F" test is poor due to small sample sizes, however, so there may be other differences in consistency that remain undetected.

Obtaining average relative potencies is an approach that appeals to some investigators instead of attempting to select the best study on which to base the relative potency estimates. To illustrate this approach, the geometric means of the potencies obtained for each PAH using the three different

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TABLE V-1

ESTIMATES OF RELATIVE POTENCY (ONE-HIT MODEL)

Experimental Study	B[a]P	B[b]F	B[j]F	CP	B[k]F	IND	B[ghi]P	D[ah]A	B[a]A	CN	B[mno]F
Deutsch-Wenzel et al. (1983) (IV-2)[b]	1[a]	0.0929			0.0536	0.146	0.0123				
LaVoie et al. (1982) (IV-3)	1	0.619[a]	0.1430[a]		0.0258						
Habs et al. (1980) (IV-6)	1[a]	0.080[a]	0.021	0.008	0.0177	0.018					
Pfeiffer (1977) (IV-7)	1[a]							1.17[a]			
Bingham and Falk (1969) (IV-9)	1								0.0101		
Van Duuren et al. (1966) (IV-11)	1	<0.001								<0.001	<0.001
Hoffmann & Wynder (1966) (IV-13)	1					0.0606[a]	0.00980				
Hoffmann & Wynder (1966) (IV-14)	1					0.0736	0.00517				
Wynder & Hoffmann (1959) (IV-18)	1							1.046[a]		0.00270	
Bryan & Shimkin (1943) (IV-19)	1							0.456[a]			

[a] Without high dose group.

[b] The table numbers in which the bioassay data and relative potency estimates derived using the two-stage model for each bioassay are shown in parentheses.

Abbreviations: B[a]P, benzo[a]pyrene; B[b]F, benzo[b]fluoranthene; B[j]F, benzo[j]fluoranthene; CP, cyclopentadieno[c,d]pyrene; B[k]F, benzo[k]fluoranthene; IND, indeno[c,d]pyrene; B[ghi]P, benzo[ghi]perylene; D[ah]A, dibenz[ah]anthracene; B[a]A, benz[a]anthracene; CN, chrysene; B[mno]F, benzo[mno]fluoranthene.

TABLE V-2

ESTIMATES OF RELATIVE POTENCY (MULTISTAGE MODEL)

Experimental Study	B[a]P	B[b]F	B[j]F	CP	B[k]F	IND	B[ghi]P	D[ah]A	B[a]A	CH	B[mno]F
Deutsch-Wenzel et al. (1983) (IV-2)[c]	1[a]	0.105			0.085	0.246	0.025[a]				
LaVoie et al. (1982) (IV-3)	1	0.562[a]	0.134[a]		0.0238						
Habs et al. (1980) (IV-6)	1[a]	0.201[a]	0.0648	0.0268	0.0279	0.0302					
Pfeiffer (1977) (IV-7)	1[a]							1.17[a]			
Bingham and Falk (1969) (IV-9)	1								0.0137		
Van Duuren et al. (1966) (IV-11)	1	<0.001								<0.001	<0.001
Hoffman & Wynder (1966) (IV-13)	1					0.0292[a]	0.0250				
Hoffman & Wynder (1966) (IV-14)	1					0.131	0.0665				
Wynder & Hoffman (1959) (IV-18)	1							4.05[a]		0.0132	
Bryan & Shimkin (1943) (IV-19)	1							0.488[b]			

[a] Without high dose group.

[b] Did not substantially change after dropping high dose group.

[c] The table numbers in which the bioassay data and relative potency estimates derived using the two-stage model for each bioassay are shown in parentheses.

Abbreviations: see Table V-1.

TABLE V-3

VARIANCE OF LOG_{10} RELATIVE POTENCY BY AGENT AND ESTIMATION METHOD

PAH	One-Hit	Multistage	Two-Stage
B[b]F	0.2456	0.1351	0.0224[a]
B[k]F	0.0598	0.0905	0.0759
B[ghi]P	0.0381	0.0602	0.0952
IND	0.1433	0.2161	0.2294
D[ah]A	0.0500	0.2133	0.0956

[a] Significantly different from the estimate obtained using the one-hit model at the $p = 0.05$ level using a standard two-sided "F" test:

$$F(3,3) = 9.28 < \frac{0.2456}{0.0224} = 10.96$$

Abbreviations: see Table V-1

models are shown in Table V-4 for all PAHs that have three or more independent estimates. Also displayed are the geometric means of each PAH among methods of estimation and for methods of estimation among PAHs. The geometric means for the same estimation methods indicate that on average the three approaches yield comparable results, with the two-stage method slightly more conservative (i.e., yields a higher estimate of risk). Selecting what is considered to be the most reliable study apparently yields results that tend to be more conservative than combining estimates.

An additional comparison of relative potencies from the "best" studies (see Section IV) using the three estimation methods is shown in Table V-5. For these estimates, the two-stage approach always gives higher relative potencies than the one-hit method and is close to or greater than that obtained using the multistage method, except for D[a,h]A. This comparison amplifies the assertions that the general two-stage approach is conservative and that selective biases to minimize risk have not been introduced by subjective handling of data.

The effects of the various approaches and assumptions used to obtain the linear terms for the B[a]P dose response models are shown in Table V-6. For ingestion exposure, the two-stage model allows for more curvature and is a point estimate of risk so a lower value is obtained than from the other models as is shown in the second line of Table V-6. The use of a data subset with more consistent exposure and duration of follow up actually increases the upper bound linear term

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TABLE V-4

GEOMETRIC MEANS OF RELATIVE POTENCIES BY AGENT AND ESTIMATION METHOD

PAH	One-Hit	Multistage	Two-Stage	Total	"Best" Study[a] (Two-Stage)
B[b]F	0.166	0.228	0.168	0.185	0.140
B[k]F	0.0290	0.0384	0.0321	0.0329	0.066
B[ghi]P	0.0085	0.0346	0.0266	0.0199	0.022
IND	0.0585	0.0729	0.1094	0.0776	0.232
D[ah]A	0.823	1.323	2.415	1.380	1.110
Total	0.0723	0.1239	0.1305	0.1053	0.1392

[a] Recommended relative potency

Abbreviations: see Table V-1

TABLE V-5

COMPARISON OF RELATIVE POTENCY ESTIMATES DERIVED FROM THE "BEST" STUDIES USING THE TWO-STAGE, ONE-HIT, AND MULTISTAGE LINEAR 95% UPPER BOUND METHODS

PAH	Two-Stage	One-Hit	95% Upper Bound
B[a]P	1.0	1.0	1.0
B[a]A	0.145	0.010	0.014
B[b]F	0.140	0.093	0.105
B[k]F	0.066	0.054	0.085
B[ghi]P	0.022	0.012	0.025
CH	0.0044	0.0023	0
D[ah]A	1.11	1.046	4.05
IND	0.232	0.146	0.246

Abbreviations: see Table V-1

TABLE V-6

EFFECT OF APPROACHES AND ASSUMPTIONS ON ESTIMATES OF THE LINEAR TERM IN B[a]P DOSE RESPONSE MODELS

		Linear Term $[mg/kg]^{-1}$		
Route of Exposure	Modification	One-Hit (point:B_1)	Multistage (95%) (upper bound:q_1)	Two-Stage (point:2AS)
Ingestion	Non-homogeneous length of exposure and follow-up data used		11.53[b]	
	Homogeneous length of exposure and follow-up data used	14.88	13.04	5.74[a]
Inhalation	Equivalency in air			
	High Dose omitted	0.917	0.789	0.301
	All data included			0.453[a]
	Additional surface area correction			
	High dose omitted		6.11[b]	

[a] Values derived in this report

[b] EPA contractor's estimates

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from 11.53 to 13.04. Using a different data set from that used by the EPA's contractor is alone thus not decreasing risk. The two-stage approach using the more homogeneous data yields a 50% overall reduction in the value obtained by EPA's contractor.

For inhalation exposure, the curvature of the dose response relationship and use of point estimates give a 62% reduction, 0.789 to 0.301, using the two-stage model as compared to the upper bound approach. However, including the high dose term and adjusting for duration of survival increase the two-stage model estimate by 50%, from 0.301 to 0.453. The major numerical difference between the methods stems from the assumptions concerning what constitutes equivalent exposure levels between species. The EPA contractor applied an additional surface area correction to the data by expressing the slope in mg/kg units. This approach is questionable and increases the term by a factor of 3 $\sqrt{70/0.05} = 7.75$, from 0.789 to 6.11. The reduction in potency after all adjustments in the data and method have been made is 93% when the method described in this report is used as compared to that derived by EPA's contractor, changing from 6.11 to 0.453.

VI. VALIDATION OF THE COMPARATIVE POTENCY APPROACH

This chapter describes how the appropriateness of the two-stage model and the accuracy of the comparative potency estimates for PAHs can be partially validated using experimental data from the literature. Studies that used combinations of PAHs to induce cancer in laboratory animals can be used to show how tumor rates predicted by the two-stage model and comparative potencies developed in this report compare to the actual tumor rates observed in the experiments. The criteria for a useful study for such a comparison are discussed below, and a study in which PAHs were tested for carcinogenesis using mice is evaluated. The model predicts tumor rates reasonably well in this experiment at lower doses, where deviations due to biological interactions are less likely.

An unbiased, sensitive, independent validation of the predictions of the low-dose additive two-stage or any other low dose extrapolation model is not possible using currently available experimental techniques. The tumor rates predicted by the model in the low dose range are far below those that can be practically measured using animal bioassays. As a result, direct experimental evidence for the model must be obtained at exposure levels far in excess of those expected in the environment. Use of high experimental levels of exposure, however, can lead to biological interactions that would not be expected to occur at environmental levels.

Among the more important interaction mechanisms anticipated at high doses are exposure saturation and enzyme saturation. In the first case, PAHs are applied to mouse skin at such high levels that they accumulate on the surface and are absorbed at relative rates that are lower than that at lower doses. As a result, higher doses administered to the animal are proportionally greater than that which is actually absorbed and predicted to reach target sites, leading to overpredictions of tumor risk at high doses. In the second case, if the levels of metabolizing enzymes are insufficient to convert all of the carcinogenic PAHs in a mixture to their reactive derivatives, the number of reactive derivatives will be fewer when PAHs are administered together as a mixture than would be predicted for the sum of the PAHs had they been administered separately; tumor rates would consequently be less than those predicted by the hypothesis of dose-additivity.

An additional factor that makes direct verification of the predictions of the model difficult is that no experiments using combinations of the specific chemicals used to evaluate potential health risks at hazardous waste sites have been conducted. As a result, validation is dependent upon the combined exposure experiments that are available in the literature. Such experiments do not include all the PAHs anticipated at hazardous waste sites such as former manufactured gas plants. In spite of these limitations, using the low-dose additive two-stage model and relative potency estimates to predict a carcinogenic response to multiple PAHs can provide partial verification of the approach.

A useful exercise in terms of validating both the comparative potency method as well as the indicator chemical approach to estimating risks at hazardous waste sites would be to use these approaches to estimate the cancer risks of some environmental complex mixtures for which bioassay and detailed analytical data are available. Appropriate information may exist for mixtures such as combustion products, coal tar extract, and cigarette smoke condensate. Such testing may reveal that the toxicity of such mixtures is partly due to components other than those commonly considered, which should therefore be included.

1. Criteria for Useful Combined Exposure Studies

For a study to provide a useful evaluation of the comparative potency method, two essential elements are required. First, B[a]P must have been included in the assay and have demonstrated a dose response relationship. Second, two or more PAHs must have been tested simultaneously. In addition, an experiment in which high-dose interaction did not affect the tumor response is desirable. Results where a quadratic dose-response relationship exists up to the highest response levels demonstrate the absence of high-dose interaction under the assumed model. Evaluating an experiment in which high-dose interaction occurred would require additional information and assumptions concerning the nature of

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the mechanisms of interaction. Sufficiently detailed biological information with which to postulate such mechanisms of interaction are not likely to be available. In this situation, an empirical (non-biologically based) dose-response modeling approach may be appropriate as a first approximation.

One study has been identified that contains sufficient information to allow an attempt to verify the basic model. This experiment is discussed and evaluated in the next section.

2. Carcinogenicity of Combined PAHs

Schmahl et al. (1977) investigated the carcinogenicity of combinations of PAHs as part of a study to evaluate automobile exhaust. Eleven PAHs were selected (not all carcinogens), combined in proportions thought to reflect those in automobile exhaust, and applied twice weekly to the backs of NMRI mice. Animals were killed if a carcinoma appeared at the site of application, or were observed until their natural deaths. Table VI-1 shows the PAHs tested and their relative proportions by weight in the mixtures. Table VI-2 shows the results of the experiments. The tumor rates observed in the experiments can be compared to those predicted using the comparative potency method. In order to do so, a dose-response relationship for B[a]P is derived in the next section and used to predict the response to a mixture given its exposure in units equivalent to B[a]P.

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TABLE VI-1

TREATMENT GROUPS FOR CARCINOGENESIS EXPERIMENT USING COMBINED PAHs

	Dose (µg)			
Control: Acetone	as solvent			
Benzo[a]pyrene	1.0		1.7	3.0
Mixture 1:				
Benzo[a]pyrene	1.0		1.7	3.0
Dibenz[a,h]anthracene	0.7		1.2	2.1
Benz[a]anthracene	1.4		2.4	4.2
Benzo[b]fluoranthene	0.9		1.5	2.7
Total	4.0		6.8	12.0
Mixture 2:				
Phenanthrene	27.0	81.0	243.0	729.0
Anthracene	8.5	25.5	76.5	229.5
Fluoranthene	10.8	32.4	97.2	291.6
Pyrene	13.8	41.4	124.2	372.6
Chrysene	1.2	3.6	10.8	32.4
Benzo[e]pyrene	0.6	1.8	5.4	16.2
Benzo[ghi]perylene	3.1	9.3	27.9	83.7
Total	65.0	195.0	585.0	1755.0

SOURCE: Schmahl et al. (1977).

TABLE VI-2

RESULTS OF CARCINOGENESIS EXPERIMENT USING COMBINED PAHs

Application	Dose (µg)[a]	Number of Animals With Carcinoma/ Effective Number of Animals		
		Observed		Predicted
Control	--	0/81		0.5
Benzo[a]pyrene	1.0	10/77	(13%)	9.0[b]
	1.7	25/88	(28%)	22.7
	3.0	43/81	(53%)	45.2
Mixture 1	4.0	25/81	(31%)	28.5
	6.8	53/88	(60%)	59.6
	12.0	63/90	(70%)	86.7[c]
Mixture 2	65.0	1/85	(1%)	0.8
	195.0	0/84		1.6
	585.0	1/88	(1%)	5.6
	1755.0	15/86	(17%)	27.8

[a] This amount was applied twice weekly to the backs of NMRI mice.

[b] $P(x)=1-\exp-[.006116(1+3.52x)^2]$

[c] Significant deviation from expected at $p = 0.01$ using X^2 goodness-of-fit test

Source: Schmahl et al. (1977)

3. Derivation of the B[a]P Dose-Response Model

In Section III of this report the following quadratic form of the dose-response model for B[a]P was derived:

$$P(x) = 1\text{-exp } [-A(1+Sx)^2],$$

where A is the background transition rate parameter and S is the exposure-related transition rate parameter. The control data from the Schmahl et al. (1977) study were used to estimate A, employing the Bayesian procedure discussed previously. Given a tumor rate of 0/80 in the control group, the Bayesian estimate of A was obtained from the following relationship:

$$(.5)/(80+1) = 1\text{-exp-}A \text{ or } A = 0.006116.$$

The parameter S was estimated using an approximate likelihood method that yielded a relative transition rate of S = 3.52. The dose-response model thus has the numerical form:

$$P(x) = 1\text{-exp-}[0.006116(1+3.52x)^2]. \qquad \text{(VI-1)}$$

The predicted number of carcinoma-bearing animals was obtained by multiplying the tumor rate obtained from equation (VI-1) times the effective number of animals in each dose group. The results of these calculations are shown in the last column of Table VI-2. A standard X^2 goodness-of-fit test yielded the result $X^2 = 0.677$ with 4-2 = 2 degrees of freedom, which has

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a "p" value of 0.73 associated with it and therefore indicates an adequate fit.

Since the quadratic model fits the B[a]P data well in the observed dose range of 0 to 3 μg, high-dose interactive effects are not expected to be important factors in that range. The predictions that are the most consistent with low-dose additivity would therefore be for mixtures composed of PAHs with a combined dose equivalent to 3 ng of B[a]P or less.

4. Predictions of Tumor Rates due to Exposure to PAH Mixtures

In order to predict the tumor rates expected as a result of exposure to combined PAHs, the potencies of the PAHs in each mixture in the Schmahl et al. (1977) study were calculated in terms of B[a]P equivalents; these are shown in Table VI-3. The lowest experimental dose was used for the calculations, as well as the relative potencies obtained in Section IV. Since the relative proportions of PAHs in each mixture were constant for all exposure levels, the B[a]P equivalent units at higher exposure levels were obtained by simple proportional multiplication. These B[a]P exposure units were substituted into equation V-1 and multiplied by the effective number of animals in each dose group to obtain the predicted number of animals with carcinomas. The predicted numbers were then compared to the observed numbers, which are shown graphically in Figure VI-1.

TABLE VI-3

CALCULATIONS OF B[a]P EQUIVALENT EXPOSURE UNITS

Dose		Relative Potency[a]	Dose (μg)	Equivalent B[a]P
Mixture 1				
Benzo[a]pyrene		1.000	1.0	1.000
Dibenz[a,h]anthracene		1.110	0.7	0.777
Benzo[a]anthracene		0.145	1.4	0.203
Benzo[b]fluoranthene		0.140	0.9	0.126
	Total		4.0	2.106
Mixture 2				
Phenanthrene[b]		0.0	27.0	0.0
Anthracene[b]		0.0	8.5	0.0
Fluoranthene[b]		0.0	10.8	0.0
Pyrene[b]		0.0	13.8	0.0
Chrysene		0.0044	1.2	0.00528
Benzo[e]pyrene[b]		0.0	0.6	0.0
Benzo[ghi]perylene		0.022	3.1	0.0682
	Total		65.0	0.07348

NOTE: Doses equivalent to B[a]P for other exposure levels were obtained by using simple ratios:

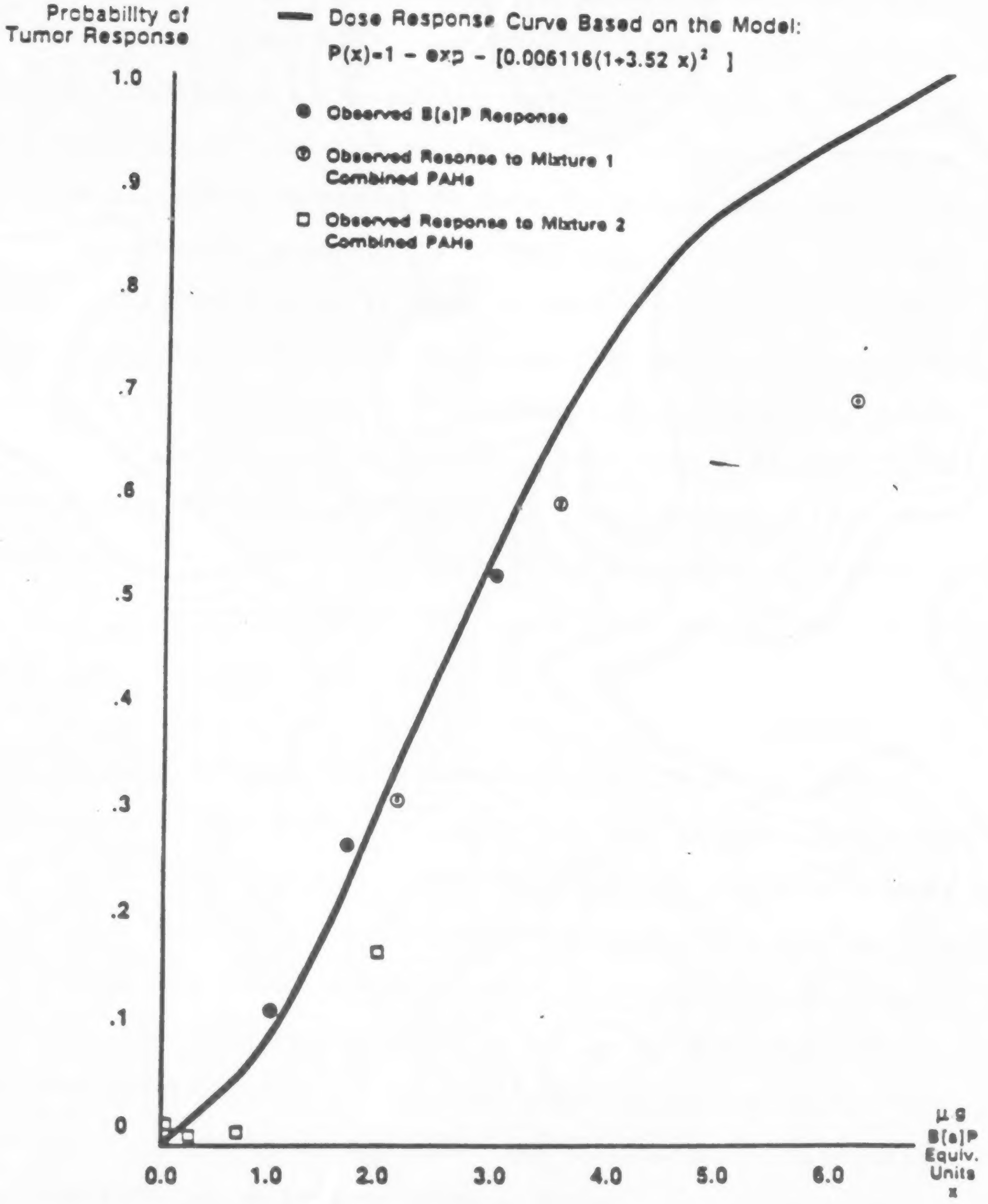
	Dose (μg)	Equivalent B[a]P Dose
Mixture 1	4.0	2.106
	6.8	3.580
	12.0	6.318
Mixture 2	65.0	0.07348
	195.0	0.22044
	585.0	0.66132
	1755.0	1.98396

[a] From Tale IV-26

[b] Considered noncarcinogenic by IARC (1983).

FIGURE VI-1

COMPARISON OF OBSERVED[a] AND PREDICTED TUMOR RATES FOR MICE EXPOSED TO PAH MIXTURES



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[a] Observed tumor rates are based on Schmahl et al. (1977).

The following observations can be made from the information given in Table VI-2. The two lower Mixture 1 exposure levels yielded predictions that were slightly larger but statistically consistent with the observed numbers of tumor-bearing animals. The highest dose led to an overprediction of the observed number by a significant amount. However, exposure at this level was more than twice that of the dose range used for B[a]P when administered alone and as a result is likely to have led to a high-dose interaction phenomenon. The relevance of this value to low-dose extrapolation is therefore questionable. For Mixture 2, the predicted values were greater than the observed values. This result may have been due to high-dose interactions or, more probably, overestimation of the potency of benzo[ghi]perylene. It should be emphasized that the predicted values were obtained without using the observed values in any manner.

5. Summary

Schmahl et al. (1977) tested B[a]P and two mixtures of both carcinogenic and noncarcinogenic PAHs for carcinogenicity when applied to the backs of mice. For each dose-group it was possible to predict tumor rates without using the experimental data from this specific study in any manner. The method yielded good estimates for combinations of the carcinogens benzo[a]pyrene, dibenz[a,h]anthracene, benz[a]anthracene, and benzo[b]fluoranthene in the same dose range observed for B[a]P alone (Mixture 1). Using potency estimates for chrysene and benzo[ghi]perylene, the effects of the agents in Mixture 2 (weak carcinogens or noncarcinogens) were systematically overestimated. In general, the method tends to overestimate at high exposure combinations and give good results at more moderate levels. As a result, the approach may be viewed as conservative for the mixtures evaluated, which in turn enhances its overall credibility.

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APPENDIX A

This appendix contains a table that displays the maximum likelihood parameter estimates, the 95% upper bounds on the linear terms, and X^2 goodness-of-fit test values for the multistage and one-hit models, respectively, along with p values that indicate how well the models fit the observed tumor data for each of the PAH carcinogenesis bioassays evaluated in Sections III and IV.

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APPENDIX A

ESTIMATES OF PARAMETERS DERIVED FROM A MULTISTAGE OR ONE-HIT MODEL

Study PAH Model	Number of Dose Groups	Maximum Likelihood Parameter Estimates								
		q0	q1	q2	q3	q4	qn	q1*	x2	p-value
Neal and Rigdon (1967)										
BaP										
Multistage (MS)	6	0	0	0	0	0	1.13E-05	7.23E-03	1.05	0.90
One-hit (OH)	6	0	8.25E-03					1.59E-02	4.28	0.37
Thyssen et al. (1981)										
BaP										
Multistage	3	0	0	4.40E-03				2.96E-02	0.62	0.43
One-hit	3	0	3.44E-02					5.71E-02	2.71	0.10
Deutsch-Wenzel et al. (1983)										
BaP										
Multistage	4	0	2.07E+00	1.01E+00	0			3.21E+00	2.7	0.10
Multistage	3a	0	2.93E-01	9.20E+00				2.29E+00	<0.001	-1
One-hit	4	0	2.60E+00					3.33E+00	3.23	0.20
One-hit	3a	0	2.44E+00					3.35E+00	3.08	0.08
B[b]F										
Multistage	4	0	0	2.74E-01	2.42E-02			2.41E-01	0.12	0.73
One-hit	4	0	2.26E-01					3.66E-01	2.23	0.33
B[k]F										
Multistage	4	0	8.43E-02	1.43E-02	0			1.89E-01	0.67	0.41
One-hit	4	0	1.30E-01					1.95E-01	0.83	0.66
IND										
Multistage	4	6.77E-03	2.49E-01	0	0			3.40E-01	2.17	0.34
Multistage	3a	0	3.56E-01	0				5.63E-01	0.75	0.39
One-hit	4	6.77E-03	2.50E-01	0				3.40E-01	2.16	0.34
One-hit	3a	0	3.56E-01	0				5.63E-01	0.75	0.39
B[ghi]P										
Multistage	4	0	2.77E-02	6.45E-04	0			5.77E-02	0.2	0.65
One-hit	4	0	3.00E-02					5.77E-02	0.19	0.91

APPENDIX A (continued)

Study PAH Model	Number of Dose Groups	q0	q1	q2	q3	q4	qn	q1*	x2	p-value
		Maximum Likelihood Parameter Estimates								
LaVoie et al. (1982)										
B[a]P										
Multistage	2	0	6.32E-02					9.90E-02	<0.001	-1
One-hit	2	0	6.32E-02					9.90E-02	<0.001	-1
B[b]F										
Multistage	4	0	2.57E-02	0	0			3.44E-02	10.3	0.06
Multistage	3a	0	3.91E-02	0				5.56E-02	2.23	0.14
One-hit	4	0	2.57E-02					3.44E-02	10.3	0.06
One-hit	3a	0	3.91E-02					5.56E-02	2.23	0.14
B[j]F										
Multistage	4	3.05E-03	5.91E-03	0	0			8.60E-03	2.01	<0.001
Multistage	3a	0	9.07E-03	0				1.33E-02	0.6	0.44
One-hit	4	3.05E-03	5.91E-03					8.60E-03	2.01	<0.001
One-hit	3a	0	9.07E-03					1.33E-02	0.6	0.44
B[k]F										
Multistage	4	0	1.63E-03	0	0			2.36E-03	1.93	0.38
One-hit	4	0	1.63E-03					2.36E-03	1.93	0.38
Habs et al. (1980)										
B[a]P										
Multistage	4	0	2.48E-01	0	0			3.11E-01	7.36	0.25
Multistage	3a	0	0	1.28E-01				1.93E-01	1.28	0.26
One-hit	4	0	2.49E-01					3.12E-01	7.37	0.025
One-hit	3a	0	2.90E-01					3.86E-01	5.77	0.016
B[b]F										
Multistage	4		3.28E-03	0	9.34E-04			2.66E-02	0.14	0.71
Multistage	3a	0	0	4.95E-03				3.87E-02	<0.001	0.92
One-hit	4	0	4.32E-02					6.74E-02	9.56	0.008
One-hit	3a	0	2.32E-02					4.08E-02	0.49	0.48
B[j]F										
Multistage	4	0	6.05E-03	0	0			1.25E-02	0.09	0.95
One-hit	4	0	6.05E-03					1.25E-02	0.09	0.95

APPENDIX A (continued)

Study PAH Model	Number of Dose Groups	Maximum Likelihood Parameter Estimates								
		q0	q1	q2	q3	q4	qn	q1*	x2	p-value
Habs et al. (1980)										
CP										
Multistage	4	0	0	0	4.03E-06			4.16E-03	0.005	0.98
One-hit	4	0	2.33E-03					5.29E-03	0.9	0.64
B[k]F										
Multistage	4	5.14E-03	0	0	0			5.17E-03	4.02	0.26
One-hit	4	5.14E-03	0					5.17E-03	4.02	0.26
BD										
Multistage	4	5.28E-03	0	0	0			5.30E-03	4.3	0.23
One-hit	4	5.28E-03	0					5.30E-03	4.3	0.23
Pfeiffer (1977)										
B[a]P										
Multistage	7	8.19E-02	2.68E-02	0	0	0	0(q5+q6)	3.02E-02	46.1	<0.001
Multistage	6a	7.42E-02	3.39E-02	0	0	0	0(q5)	3.86E-02	18.3	0.001
One-hit	7	8.18E-02	2.68E-02					3.02E-02	46.1	<0.001
One-hit	6a	7.42E-02	3.39E-02					3.86E-02	18.3	0.001
D[ah]A										
Multistage	7	1.27E-01	2.50E-02	0	0	0	0(q5+q6)	2.88E-02	100.4	0
Multistage	6a	1.12E-01	3.92E-02	0	0	0	0(q5)	4.53E-02	55.7	<0.001
One-hit	7	1.27E-01	2.50E-02					2.88E-02	100.4	0
One-hit	6a	1.02E-01	3.42E-02					4.54E-02	55.7	<0.001
Bingham and Falk (1969)										
B[a]P										
Multistage	5	0	0	0	0	3.37E+6		2.98E+01	0.001	-1
One-hit	5	0	2.16E+01					4.20E+01	1.21	0.75
B[a]A										
Multistage	5	1.44E-02	2.18E-01	0	0	0		4.10E-01	3.17	0.37
One-hit	5	1.44E-02	2.18E-01					4.10E-01	3.17	0.37

APPENDIX A (continued)

Study PAH Model	Number of Dose Groups	q0	q1	q2	q3	q4	qn	q1*	x2	p-value
VanDuuren et al. (1966)										
B[a]P										
Multistage	2	2.88E-01	1.69E+02					4.86E+19	<0.001	-1
One-hit	2	2.88E-01	1.69E+02					4.86E+19	<0.001	-1
CH										
Multistage	2	2.88E-01	1.32					2.23	<0.001	-1
One-hit	2	2.88E-01	1.32					2.23	<0.001	-1
B[b]F										
Multistage	2	2.88E-01	2.01					3.43	<0.001	-1
One-hit	2	2.88E-01	2.01					3.43	<0.001	-1
B[mno]F										
Multistage	2	2.55E-01	0					2.37E-01	0.14	0.70
One-hit	2	2.55E-01	0					2.37E-01	0.14	0.70
Hoffman and Wynder (1966)										
B[a]P										
Multistage	3	0	3.43E+01	0				4.90E+01	0.32	0.57
One-hit	3	0	3.43E+01	0				4.90E+01	0.32	0.57
DB										
Multistage	5	0	1.18	0	0	0		1.82	9.36	0.025
Multistage	4	0	0	0	3.10E+02			1.43	0.911	0.634
One-hit	5	0	1.18					1.82	9.36	0.025
One-hit	4	0	2.08					3.80	4.27	0.118
B[ghi]P										
Multistage	3	0	3.36E-01	0				1.23	2.04	0.15
One-hit	3	0	3.36E-01					1.23	2.04	0.15
Hoffman and Wynder (1966)										
B[a]P										
Multistage	2	6.90E-02	6.16					8.97	<0.001	-1
One-hit	2	6.90E-02	6.16					8.97	<0.001	-1

APPENDIX A (continued)

Study PAH Model	Number of Dose Groups	q0	q1	q2	q3	q4	qn	q1*	x2	p-value
		Maximum Likelihood Parameter Estimates								
Hoffman and Wynder (1966)										
ND										
Multistage	2	6.90E-02	4.53E-01					1.18	<0.001	-1
One-hit	2	6.90E-02	4.53E-01					1.18	<0.001	-1
B[ghi]P										
Multistage	2	6.90E-02	3.19E-02					5.97E-01	<0.001	-1
One-hit	2	6.90E-02	3.19E-02					5.97E-01	<0.001	-1
Wynder and Hoffman (1959)										
B[a]P										
Multistage	3	0	0	3.76E+04				5.83E+01	1.18	0.277
Multistage	4	0	0	3.47E+04	0			6.61E+01	1.61	0.45
One-hit	3	0	1.51E+02					2.18E+02	6.01	0.014
One-hit	4	0	1.89E+02					2.51E+02	7.69	0.021
D[ah]A										
Multistage	3	0	9.15E+01	1.39E+04				2.68E+02	<0.001	-1
One-hit	3	0	1.98E+02					2.98E+02	1.1	0.29
CH										
Multistage	2	0	5.11E-01					8.75E-01	<0.001	-1
One-hit	2	0	5.11E-01					8.75E-01	<0.001	-1
Bryan and Shimkin (1943)										
B[a]P										
Multistage	13	2.61E-02	1.72	0	0	0	0(q5-q12)=0	2.11	4.79E+03	<0.001
One-hit	13	2.61E-02	1.72	0				2.11	4.79E+03	<0.001
D[ah]A										
Multistage	13	0	7.87E-01					1.53	1073	<0.001
One-hit	13	2.45E-01	7.87E-01					1.03	1073	<0.001

b Highest dose group omitted.

APPENDIX B

This appendix contains a table for each of the PAH carcinogenesis bioassays evaluated in Sections III and IV of this report that shows the doses of PAHs administered, numbers of tumor-bearing animals observed, and the numbers of tumor-bearing animals that were predicted from the relative potencies derived using the multistage or one-hit models.

Table B-1

Data Used to Estimate the Dose-Response Relationship Between Ingested B[a]P and Forestomach Tumors

PAH	Dose (mg/kg/day)	Number of Mice Exposed	Number of Tumor Bearing Animals		
			Observed	Predicted	
				Multistage	One-Hit
B[a]P[a]	0	289	0	0	0
	0.13	25	0	<0.001	0.027
	1.30	24	0	0.001	0.256
	3.90	37	0	0.374	1.17
	5.20	40	1	1.68	1.68
	5.85	40	4	2.97	1.88

---

[a] Abbreviations: B[a]P, benzo[a]pyrene.

Source: Neal and Rig[illegible]on (1967).

Table B-2

Data Used To Estimate the Dose-Response Relationship Between B[a]P and Respiratory Tract Tumors

PAH	Exposure Rate (mg B[a]P/m^3)	Number of Hamsters Exposed	Number of Tumor Bearing Animals Observed	Predicted Multistage	One-Hit
B[a]P[a]	0	27	0	0	0
	2.2	27	0	0.569	1.97
	9.5	26	9	8.53	7.26
	46.5	25	(b)		

---

[a] Abbreviations: B[a]P, benzo[a]pyrene.

[b] Highest dose group omitted because of lack of fit.

Source: Thyssen et al. (1981)

Table B-3

Data Used to Estimate the Dose-Response Relationship Between Injected (lung) B[a]P and Selected PAHs and Epidermoid Carcinomas

PAH	Exposure Levels in mg	Number of Rats Exposed	Number of Tumor Bearing Animals Observed	Predicted Multistage	Predicted One-Hit
B[a]P[a]	0	35	0	0	0
	0.1	35	4	4	7.57
	0.3	35	21	21	18.15
	1.0	35	(b)		
B[b]F	0	35	0	0	0
	0.1	35	0	0.097	0.783
	0.3	35	1	0.875	2.30
	1.0	35	9	9.03	7.09
B[k]F	0	35	0	0	0
	0.16	35	0	0.482	0.723
	0.83	31	3	2.38	3.18
	4.15	27	12	12.12	11.29
IND	0	35	0	0	0
	0.16	35	3	1.94	1.94
	0.83	35	8	8.95	8.95
	4.15	35	(b)		
B[ghi]P	0	35	0	0	0
	0.16	35	0	0.156	0.167
	0.83	35	1	0.812	0.860
	4.15	34	4	4.03	3.98

---

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[b]F, benzo[b]fluoranthene; B[k]F benzo[k]fluoranthene; IND, indeno(1,2,3-cd)pyrene; B[ghi]P, benzo[ghi]perylene

[b] Highest dose group omitted from analysis because of lack of fit.

Source: Deutch-Wenzel et al. (1983).

Table B-4

Data Used to Estimate the Dose-Response Relationship Between Skin Painted B[a]P and Selected PAHs and Squamous Cell Papilloma

PAH	Total Initiating Dose (μg)	Number of Mice Exposed	Number of Tumor Bearing Animals: Observed	Predicted: Multistage	Predicted: One-Hit
B[b]F[a]	0	20	0	0	0
	10	20	9	6.47	6.47
	30	20	12	13.80	13.80
	100	20	(b)		
B[j]F	0	20	0	0	0
	30	20	6	4.77	4.77
	100	20	11	11.93	11.93
	1000	20	(b)		
B[k]F	0	20	0	0	0
	30	20	1	0.955	0.955
	100	20	5	3.01	3.01
	1000	20	15	16.09	16.09
B[a]P	0	20	0	0	0
	30	20	17	17	17

---

[a] Abbreviations: B[b]F, benzo[b]fluoranthene; B[j]F, benzo[j]fluoranthene; B[k]F, benzo[k]fluoranthene; B[a]P, benzo[a]pyrene

[b] Highest dose group omitted from analysis because of lack of fit.

Source: LaVoie et al. (1982).

Table B-5

Data Used to Estimate the Dose-Response Relationship Between Skin-Painted B[a]P and Selected PAHs and Tumors

PAH	PAH Dose (pg/animal)	Number of Mice Exposed	Number of Tumor Bearing Animals		
			Observed	Predicted	
				Multistage	One-Hit
B[a]P[a]	0	80	0	0	0
	1.7	34	8	10.48	13.23
	2.8	35	24	22.12	19.46
	4.6	36	(b)		
B[b]F	0	80	0	0	0
	3.4	38	2	2.11	2.88
	5.6	34	5	4.89	4.14
	9.2	37	(b)		
B[j]F	0	80	0	0	0
	3.4	38	1	0.774	0.774
	5.6	35	1	1.17	1.17
	9.2	38	2	2.08	2.06
CP	0	80	0	0	0
	1.7	34	0	0.001	0.134
	6.8	35	0	0.044	0.550
	27.2	38	3	2.96	2.33
B[k]F	0	80	0	0.410	0.410
	3.4	39	1	0.200	0.200
	5.6	38	0	0.195	0.195
	9.2	38	0	0.195	0.195
IP	0	80	0	0.421	0.421
	3.4	36	1	0.189	0.189
	5.6	37	0	0.195	0.195
	9.2	37	0	0.195	0.195

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[b]F, benzo[b]fluoranthene; B[j]F, benzo[j]fluoranthene; CP, cyclopentadieno(cd)pyrene; B[k]F benzo[k]fluoranthene; IP, indeno(1,2,3-cd)pyrene

[b] Highest dose group omitted from analysis because of lack of fit.

Source: Habs et al. (1980).

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Table B-6

Data Used to Estimate the Dose-Response Relationship Between Injected (subcutaneous) B[a]P and D[ah]A and Sarcomas

PAH	Dose (μg)	Number of Mice Exposed	Number of Tumor Bearing Animals Observed	Predicted Multistage	Predicted One-Hit
B[a]P[a]	0	600	42	42.94	42.94
	3.12	100	9	16.47	16.47
	6.25	100	35	24.87	24.87
	12.05	100	61	39.20	39.20
	25.0	100	57	60.18	60.18
	50.0	100	77	82.92	82.92
	100.0	100	(b)		
D[ah]A	0	600	42	58.04	58.04
	2.35	100	37	17.82	17.62
	4.7	100	39	24.87	24.87
	9.3	100	44	37.27	37.27
	18.7	100	56	56.60	56.60
	37.5	100	65	79.23	79.23
	75.0	100	(b)		

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[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene

[b] Highest dose group omitted from analysis because of lack of fit.

Source: Pfeiffer (1977).

Table B-7

Data used to Estimate the Dose-Response Relationship Between Skin Painted B[a]P and B[a]A and Malignant Tumors

PAH	Concentration (%)	Number of Mice Exposed	Number of Tumor Bearing Animals Observed	Predicted Multistage	Predicted One-Hit
B[a]P[a]	0	20	0	0	0
	0.00002	18	0	<0.001	0.008
	0.0002	21	0	<0.001	0.091
	0.002	20	0	0.001	0.847
	0.02	12	5	5.00	4.22
B[a]A	0	20	0	0.287	0.287
	0.002	32	0	0.473	0.473
	0.02	18	1	0.335	0.335
	0.2	32	3	1.80	1.80
	1.0	29	5	6.01	6.01

[a] Abbreviations: B[a]P, benzo[a]pyrene; B[a]A, benz[a]anthracene.

Source: Bingham and Falk (1969).

Table B-8

Data Used to Estimate the Dose-Response Relationship between Skin Painted B[a]P and Selected PAHs and Carcinomas

PAH	Dose (mg)	Number of Mice Exposed	Number of Tumor Bearing Animals		
			Observed	Predicted	
				Multistage	One-Hit
B[a]P[a]	0	20	5	5	5
	0.150	20	20	20	20
CH	0	20	5	5	5
	1	20	16	16	16
B[b]F	0	20	5	5	5
	1	20	18	18	18
B[mno]F	0	20	5	4.5	4.5
	1	20	4	4.5	4.5

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[a] Abbreviations: B[a]P, Benzo[a]pyrene; CH, Chrysene; B[b]F, Benzo[b]fluoranthene; B[mno]F, Benzo[mno]fluoranthene.

Source: Van Duuren et al. (1966).

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Table B-9

Data Used to Estimate the Dose-Response Relationship Between Skin Painted B[a]P, IP, and B[ghi] Tumors

PAH	Dose (%)	Number of Mice Exposed	Number of Tumor Bearing Animals		
			Observed	Predicted	
				Multistage	One-Hit
B[a]P[a]	0	100	0	0	0
	0.05	20	17	16.40	16.40
	0.1	20	19	19.35	19.35
IP	0	100	0	0	0
	0.01	20	0	0.006	0.411
	0.05	20	0	0.760	1.97
	0.1	20	6	5.33	3.75
	0.5	20	(b)		
B[ghi]P	0	100	0	0	0
	0.05	20	1	0.333	0.333
	0.1	20	0	0.661	0.661

[a] Abbreviations: B[a]P, benzo[a]pyrene; IP, indeno(1,2,3-cd)pyrene; B[ghi]P, benzi[ghi]perylene.

[b] Highest dose group omitted from analysis because of lack of fit.

Source: Hoffman and Wynder (1966).

Table B-10

Data Used to Estimate the Dose-Response Relationship Between Skin Painted B[a]P and Selected PAHs and Tumors

PAH	Total Dose (mg)	Number of Mice Exposed	Number of Tumor Bearing Animals		
			Observed	Predicted	
				Multistage	One-Hit
B[a]P[a]	0	30	2	2	2
	0.25	30	24	24	24
IP	0	30	2	2	2
	0.25	30	5	5	5
B[ghi]P	0	30	2	2	2
	0.25	27	2	2	2

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[a] Abbreviations: B[a]P, benzo[a]pyrene; IP, indeno(1,2,3-cd)pyrene; B[ghi]P, benzo[ghi]perylene

Source: Hoffman and Wynder (1966).

Table B-11

Data Used to Estimate the Dose-Response Relationship Between Skin Painted B[a]P and Selected PAHs and Carcinomas

PAH	Dose (% concentration)	Number of Mice Exposed	Number of Tumor Bearing Animals Observed	Predicted Multistage	Predicted One-Hit
B[a]P[a]	0	20	0	0	0
	0.001	29	0	1.07	4.07
	0.005	30	19	18.29	10.08
D[ah]A	0	20	0	0	0
	0.001	20	2	2	3.59
	0.01	20	18	18	17.24
	0.1	20	([b])		
CH	0	20	0	0	0
	1	20	8	8	8

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[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene CH, chrysene.

[b] Highest dose group omitted from analysis because of lack of fit.

Source: Wynder and Hoffman (1959).

Table B-12

Data Used to Estimate the Dose-Response Relationship Between Subcutaneously Injected B[a]P and Selected PAHs and Spindle-Cell Carcinomas

PAH	Dose (mg)	Number of Mice Exposed	Number of Tumor Bearing Animals		
			Observed	Predicted	
				Multistage	One-Hit
B[a]P[a]	0	160	4	4.12	4.12
	0.00195	81	2	2.35	2.35
	0.0078	40	0	1.55	1.55
	0.0156	19	0	.981	.981
	0.031	16	0	1.22	1.22
	0.062	20	4	2.49	2.49
	0.125	19	15	4.08	4.08
	0.25	21	14	7.70	7.70
	.5	19	19	11.18	11.18
	1.0	20	18	16.52	16.52
	2.0	19	19	18.41	18.41
	4.0	19	16	18.98	18.98
	8.0	21	20	21.00	21.00
D[ah]A	0	160	4	34.73	34.73
	0.00195	79	2	17.24	17.24
	0.0078	40	6	8.87	8.87
	0.0158	19	6	4.31	4.31
	0.031	21	16	4.95	4.95
	0.062	20	20	5.09	5.09
	0.125	23	21	6.68	6.68
	0.25	21	19	7.49	7.49
	0.5	21	20	9.91	9.91
	1.0	22	22	14.16	14.16
	2.0	19	19	15.92	15.92
	4.0	20	17	19.33	19.33
	8.0	21	16	20.97	20.97

[a] Abbreviations: B[a]P, benzo[a]pyrene; D[ah]A, dibenz[ah]anthracene

Source: Bryan and Shimkin (1943).

BENZ(A)ANTHRACENE

MEDIA	SOIL TYPE	ORGANIC MATTER	MOISTURE	TEMPERATURE	APP. RATE	INITIAL CONC	HALF-LIFE	DEGRADATION RATE UNITS	ACCLIMATION	DURATION	REFERENCE
API Separator Sludge Waste	Kidman Sandy Loam		-2 to -4 bars	10-22 C	5%	[illegible]	[illegible]	0.0006 1/DAY	1	120 days	U.S. EPA (1987)
API Separator Sludge Waste	Durant Clay Loam		-2 to -4 bars	10-22 C	5%	31.4	1100	0.0006 1/DAY	1	240 days	U.S. EPA (1987)
PAH mixture			60% cap.	20 C		5	261	0.0026 1/DAY	1	40 (days)	Park Report (1985)
PCP wood preserving wastes	Durant Clay Loam		-1 bar	10-22 C	0.75	43	1000	0.0007 1/DAY	1	100 days	U.S. EPA (1987)
Slop Oil Emulsion Solids	Durant Clay Loam		-2 to -4 bars	10-22 C	1%	20	630	0.0011 1/DAY	1	270 days	U.S. EPA (1987)
PCP wood preserving wastes	Durant Clay Loam		-2 to -4 bars	10-22 C	0.75	16.4	770	0.0009 1/DAY	1	240 days	U.S. EPA (1987)
Creosote wood preserving wastes	Durant Clay Loam		1 bar	10-22 C	1.5	43	495	0.0014 1/DAY	1	100 days	U.S. EPA (1987)
Slop Oil Emulsion Solids	Durant Clay Loam		-1 bar	10-22 C	1%	4.0	347	0.002 1/DAY	1	100 days	U.S. EPA (1987)
						20.6	231	0.003 1/DAY	1		Gardner et al. (1979)
						42.6	252	0.003 1/DAY	1		Gardner et al. (1979)
Refinery waste	Nunn Clay Loam	0.1	26 (sat.)		20.0%	311.5	231	0.003 1/DAY	1		A.P.I. (1987)
						72.8	176	0.004 1/DAY	1		Gardner et al. (1979)
Refinery waste	Nunn Clay Loam	0.1	60 (sat.)		6.0%	171.6	173	0.004 1/DAY	1		A.P.I. (1987)
Creosote wood preserving wastes	Durant Clay Loam		-2 to -4 bars	10-22 C	1.5	[illegible]	170	0.0042 1/DAY	1	280 days	U.S. EPA (1987)
						25.4	176	0.004 1/DAY	1		Gardner et al. (1979)
Pure compound						0.15	154	0.0045 1/DAY	1		Sims (1982)
Refinery waste	Nunn Clay Loam	0.1	26 (sat.)		20.0%	45.8	170	0.005 1/DAY	1		A.P.I. (1987)
Pure compound						0.1	162	0.005 1/DAY	1		Sims (1982)
Complex fossil fuel waste						91	123	0.0056 1/DAY	1 C		Sims (1984)
						22.1	119	0.0058 1/DAY	1		Gardner et al. (1979)
						1.5	102	0.0068 1/DAY	1 C		Groenewegen & Stolp (1976)
Creosote wood preserving wastes	Durant Clay Loam		-2 to -4 bars	10-22 C	0.75	2.6	94	0.0074 1/DAY	1	280 days	U.S. EPA (1987)
Refinery waste	Kidman Sandy Loam	0.1	24.52 (sat.)		65.0%	540.6	87	0.008 1/DAY	1		A.P.I. (1987)
						17.2	174	0.008 1/DAY	1		Gardner et al. (1979)
PAH mixture			60% cap.	20 C		50	130	0.005 1/DAY	1	40 (days)	Park Report (1985)
Refinery waste	Kidman Sandy Loam	0.1	24.52 (sat.)		20.0%	1.0	77	0.009 1/DAY	1		A.P.I. (1987)
Creosote wood preserving wastes	Durant Clay Loam		-2 to -4 bars	10-22 C	1.0%	4	71	0.0097 1/DAY	1	280 days	U.S. EPA (1987)
PCP wood preserving wastes	Durant Clay Loam		-1 bar	10-22 C	0.5%	29	71	0.0097 1/DAY	1	100 days	U.S. EPA (1987)
Refinery waste	Kidman Sandy Loam	0.1	24.52 (sat.)		6.0%	272	61	0.011 1/DAY	1		A.P.I. (1987)
Complex fossil fuel waste with amendments						11	52	0.0133 1/DAY	1 C		Sims (1984)
Creosote wood preserving waste	FINE SANDY LOAM	[illegible]		5-30	10 g/100g	244	50	0.0139 1/DAY	1 C	575 (days)	ReTeC (UM 1987)
Pure compound						7	43	0.016 1/DAY	1		Sims (1982)
Creosote wood preserving waste	FINE SANDY LOAM	[illegible]		5-30	10 g/100g	475	43	0.0161 1/DAY	1 C	575 (days)	ReTeC (UM 1987)
Creosote wood preserving waste	McLaurin fine sandy loam		80% field cap.	10-22		103.2	41	0.0169 1/DAY	1 C	180 (days)	ReTeC (UM 1987)
Pure compound						4.7	41	0.0169 1/DAY	1 C		Sims (1982)
Slop Oil Emulsion Solids	Durant Clay Loam		-1 bar	10-22 C	1.5%	540	27	0.026 1/DAY	1	100 days	U.S. EPA (1987)
				<15		0.12	15.2	0.046 1/DAY	1		Herbes & Schwall (1978)
Mixture of PAH in soils	SANDY LOAM	7.9	80-100 % cap.	10 C		11.1	540	0.0221 mg/kg/day	0 C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9	80-100 % cap.	20 C		11.1	430	0.0250 mg/kg/day	0 C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9	40-100 % cap.	30 C		11.1	240	0.0227 mg/kg/day	0 C	240 (days)	Coover & Sims (1987)
Creosote wood preserving waste	FINE SANDY LOAM	[illegible]		5-30	10 g/100g	475	344	0.4400 mg/kg/day	0 C	575 (days)	ReTeC (UM 1987)
Creosote wood preserving waste	FINE SANDY LOAM	[illegible]		5-30	10 g/100g	244	218	0.5200 mg/kg/day	0 C	575 (days)	ReTeC (UM 1987)
Creosote wood preserving waste	McLaurin fine sandy loam		80% field cap.	10-22		103.2	96	0.5700 mg/kg/day	0 C	180 (days)	ReTeC (UM 1987)

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[illegible]

MEDIA	SOIL TYPE	SOIL pH	[illegible]	MOISTURE	TEMPERATURE	APP. RATE	INITIAL CONC	HALF-LIFE	RATE CONSTANT UNITS		REACTION ORDER	DURATION	SOURCE
					<15		0.91	154	0.0010 1/DAY		1 C		Herles & Schmall (197[illegible]) a
					15.5		17	4.0	0.0017 1/DAY		1 C		Gardner et al. (1979) a
					<15		0.0014	4.6	0.0017 1/DAY		1 C		Shabad et al (1971) *
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	16-22 C	1.0	2	231	0.0030 1/DAY		1	200 days	U.S. EPA (1987)
		6.1			15.5		9.5	234	0.003 1/DAY		1		Gardner et al. (1979) *
							17	4.0	0.172 1/DAY		1		Gardner et al. (1979) *
Creosote wood preserving waste	McLaurin fine sandy loam				15.5		9.5	234	0.0029 1/DAY		1 C		Gardner et al. (1979) a
				80% field cap.	18-22		151.2	241	0.0029 1/DAY		1 C	180 (days)	RelaC (JH 1987)
		6.1			15.5		7.6	234	0.003 1/DAY		1		Gardner et al. (1979) *
Creosote wood preserving waste	FINE SANDY LOAM		6 [illegible]		5-30	20 g/kg/y	229	204	0.0034 1/DAY		1 C	575 (days)	RelaC (JH 1987)
					15.5		42.6	175	0.0040 1/DAY		1 C		Gardner et al. (1979) a
					15.5		42.6	175	0.004 1/DAY		1		Gardner et al. (1979) *
					15.5		12.1	147	0.0047 1/DAY		1 C		Gardner et al. (1979) a
		6.1			15.5		12.1	147	0.005 1/DAY		1		Gardner et al. (1979) *
PCP wood preserving waste	Durant Clay Loam			-2 to -4 bars	16-22 C	0.75	10.2	140	0.0050 1/DAY		1	240 days	U.S. EPA (1987)
API Separator Sludge Waste	Durant Clay Loam			-1 bar	16-22 C	95	170	105	0.0066 1/DAY		1	100 days	U.S. EPA (1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 [illegible]		5-30	10 g/kg/y	1.9	103	0.0067 1/DAY		1 C	575 (days)	RelaC (JH 1987)
Slop Oil Emulsion Solids	Durant Clay Loam			-1 bar	16-22 C	192	55	91	0.0076 1/DAY		1	100 days	U.S. EPA (1987)
Complex fossil fuel waste							80	91	0.0076 1/DAY		1 C		Sims (1986)
Complex fossil fuel waste with amendments							80	69	0.0100 1/DAY		1 C		Sims (1986)
					<15		0.545	65	0.0106 1/DAY		1 C		Shabad et al (1971) *
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	0.78	1.4	61	0.0114 1/DAY		1	240 days	U.S. EPA (1987)
					15.5		1.4	57	0.012 1/DAY		1		Groenewegen & Stolp (1976) a
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	16-22 C	1.18	1.6	54	0.0128 1/DAY		1	200 days	U.S. EPA (1987)
					<15		0.099	50	0.0139 1/DAY		1 C		Herles & Schmall (1978) a
					<15		28.5	47	0.0147 1/DAY		1 C		Shabad et al (1971) *
							10.5	30	0.0231 1/DAY		1 C		Gardner et al. (1979) *
					15.5		10.5	30	0.0231 1/DAY		1 C		Gardner et al. (1979) a
Slop Oil Emulsion Solids	Durant Clay Loam			-2 to -4 bars	16-22 C	1.25	52.0	24	0.0289 1/DAY		1	240 days	U.S. EPA (1987)
Slop Oil Emulsion Solids	Durant Clay Loam			-2 to -4 bars	18-22 C	1.95	11.3	21	0.0329 1/DAY		1	200 days	U.S. EPA (1987)
Slop Oil Emulsion Solids	Durant Clay Loam			-1 bar	18-22 C	1.75	72	14	0.0495 1/DAY		1	100 days	U.S. EPA (1987)
					15		19.5	7	0.099 1/DAY		1		Poglazova et al. (1972 c) *
Mixture of PAH in soils				80% cap.	30 C		50	210	0.1031 1/DAY	8	0	400 (days)	Pace Report (1985)
					15		170.6	6	0.116 1/DAY		1		Poglazova et al. (1967) *
					15		0.0015	5	0.1386 1/DAY		1 C		Shabad et al (1971) *
					15		19.5	5	0.139 1/DAY		1		Poglazova et al. (1972 b) *
					15		10.6	4	0.173 1/DAY		1		Poglazova et al. (1967) *
					15		19.5	3	0.231 1/DAY		1		Poglazova et al. (1972 c) *
					15		1	2	0.3466 1/DAY		1 C		Shabad et al (1971) *
					15		0.515	2	0.3466 1/DAY		1 C		Shabad et al (1971) *
					15		1	2	0.3466 1/DAY		1 C		Shabad et al (1971) a
Mixture of PAH in soils				60% cap.	30 C		5	247	0.0020 1/DAY	2	0	400 (days)	Pace Report (1985)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100% Cap.	10 C		10.4	580	0.0120 mg/kg/day		0 C	240 (days)	Grover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100% Cap.	20 C		10.2	240	0.0106 mg/kg/day		0 C	240 (days)	Grover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100% Cap.	30 C		10.2	220	0.0235 mg/kg/day		0 C	240 (days)	Grover & Sims (1987)
[illegible] wood preserving waste	FINE SANDY LOAM		6 [illegible]		5-30	10 g/kg/y	224	1540	[illegible] mg/kg/day		0 C	575 (days)	RelaC (JH 1987)
[illegible]	FINE SANDY LOAM		6 [illegible]		5-30	10 g/kg/y	1.5	[illegible]	[illegible] mg/kg/day		0 C	575 (days)	RelaC (JH 1987)
[illegible]	McLaurin fine sandy loam			80% field cap.	15-22		151.2	219	0.0452 mg/kg/day		0 C	190 (days)	RelaC (JH 1987)

BENZO(B)FLUORANTHENE

MEDIA	SOILTYPE	SOILpH	AMNDTYPE	MOISTURE	TEMPRANGE	APPLRATE	INITCONC	HALFLIFE	RTECONST	RTEUNITS	REACTORDER	DURATION	STUDYREF
Creosote wood preserving waste	McCollum fine sandy loam			30 % field cap.	18-22		106.2	174	0.0040	1/DAY	1-C	160 (days)	RelaC (JH 1987)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	1.05	1.8	1300	0.0005	1/DAY	1	320 days	U.S. EPA (1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	167	825	0.0008	1/DAY	1-C	575 (days)	RelaC (JH 1987)
API Separator Sludge Waste	Durant Clay Loam			-1 bar	18-22 C	5%	110	630	0.0011	1/DAY	1	100 days	U.S. EPA (1987)
PCP wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	0.72	12	315	0.0022	1/DAY	1	100 days	U.S. EPA (1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	112	289	0.0024	1/DAY	1-C	575 (days)	RelaC (JH 1987)
PCP wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	0.72	53	270	0.0026	1/DAY	1	240 days	U.S. EPA (1987)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	1.2%	2.1	250	0.0028	1/DAY	1	320 days	U.S. EPA (1987)
Creosote wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	1.2%	18	158	0.0044	1/DAY	1	100 days	U.S. EPA (1987)
Pure compound		6.1					0.3	130	0.0053	1/DAY	1-C		Sims (1982)
Pure compound		6.1					0.57	123	0.0056	1/DAY	1-C		Sims (1982)
Pure compound		6.1					0.13	98	0.0071	1/DAY	1-C		Sims (1982)
Pure compound		6.1					46	85	0.0082	1/DAY	1-C		Sims (1982)
Complex fossil fuel waste							21	85	0.0082	1/DAY	1-C		Sims (1986)
Pure compound		6.1					53	73	0.0095	1/DAY	1-C		Sims (1982)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	0.72	1.2	67	0.0103	1/DAY	1	280 days	U.S. EPA (1987)
Pure compound		6.1					33	67	0.0103	1/DAY	1-C		Sims (1982)
Complex fossil fuel waste with amendments							21	65	0.0107	1/DAY	1-C		Sims (1986)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100 % Cap.	20 C		9.94	610	0.0081	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100 % Cap.	10 C		9.94	530	0.0094	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100 % Cap.	30 C		9.94	340	0.0146	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Creosote wood preserving waste	McCollum fine sandy loam			30 % field cap.	18-22		106.2	1083	0.0490	mg/kg/day	0-C	160 (days)	RelaC (JH 1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	167	1210	0.0690	mg/kg/day	0-C	575 (days)	RelaC (JH 1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	112	357	0.1569	mg/kg/day	0-C	575 (days)	RelaC (JH 1987)

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CHRYSENE

MEDIA	SOILTYPE	SOILpH	AMMTYPE	MOISTURE	TEMPRANGE	APPLRATE	INITCONC	HALFLIFE	RATECONST	RCUNITS	REACTNTYPE	DURATION	STUDYREF
							4.4		NA	1/DAY	1-C		Groenewegen & Stolp (1976) •
PCP wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	0.5%	46	5000	0.0001	1/DAY	1	100 days	U.S. EPA (1987)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	1.0%	4.1	400	0.0012	1/DAY	1	280 days	U.S. EPA (1987)
API Separator Sludge Waste	Kidman Sandy Loam			-2 to -4 bars	18-22 C	6%	21.2	1200	0.0006	1/DAY	1	200 days	U.S. EPA (1987)
Slop Oil Emulsion Solids	Durant Clay Loam			-2 to -4 bars	18-22 C	14%	140	630	0.001	1/DAY	1	280 days	U.S. EPA (1987)
PCP wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	0.7%	35.1	410	0.0017	1/DAY	1	280 days	U.S. EPA (1987)
Creosote wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	1.3%	43	330	0.0021	1/DAY	1	100 days	U.S. EPA (1987)
API Separator Sludge Waste	Durant Clay Loam			-2 to -4 bars	18-22 C	6%	56	290	0.0024	1/DAY	1	280 days	U.S. EPA (1987)
PCP wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	0.7%	26	173	0.0040	1/DAY	1	100 days	U.S. EPA (1987)
Slop Oil Emulsion Solids	Durant Clay Loam			-1 bar	18-22 C	1.5%	210	161	0.0043	1/DAY	1	100 days	U.S. EPA (1987)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	1.6%	5.3	130	0.0054	1/DAY	1	280 days	U.S. EPA (1987)
Refinery waste	Nunn Clay Loam	8.1		35% (sat.)		8% ORG	67.5	116	0.006	1/DAY	1		A.P.I. (1987)
Refinery waste	Nunn Clay Loam	8.1		33% (sat.)		4% ORG	47	99	0.007	1/DAY	1		A.P.I. (1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	123	95	0.0073	1/DAY	1-C	575 (days)	RetoC (JH 1987)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	0.7%	3.1	78	0.0089	1/DAY	1	280 days	U.S. EPA (1987)
Refinery waste	Nunn Clay Loam	8.1		33% (sat.)		2% ORG	27	77	0.009	1/DAY	1		A.P.I. (1987)
Refinery waste	Kidman Sandy Loam	8.1		24.5% (sat.)		6% ORG	130.5	77	0.0090	1/DAY	1		A.P.I. (1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	357	73	0.0096	1/DAY	1-C	575 (days)	RetoC (JH 1987)
Complex fossil fuel waste							27	70	0.0099	1/DAY	1-C		Sims (1986)
Refinery waste	Kidman Sandy Loam	8.1		24.5% (sat.)		4% ORG	78	58	0.012	1/DAY	1		A.P.I. (1987)
API Separator Sludge Waste	Durant Clay Loam			-1 bar	18-22 C	5%	95	53	0.0132	1/DAY	1	100 days	U.S. EPA (1987)
Complex fossil fuel waste with amendments							27	42	0.0165	1/DAY	1-C		Sims (1986)
Refinery waste	Kidman Sandy Loam	8.1		24.5% (sat.)		2% ORG	40	41	0.017	1/DAY	1		A.P.I. (1987)
Creosote wood preserving waste	McCollum fine sandy loam			80% field cap.	13-22		106.2	40	0.0173	1/DAY	1-C	180 (days)	RetoC (JH 1987)
					15-25		500	10.5	0.0660	1/DAY	1-C		Medvedev & Davidov (1972) •
					15-25		5	5.5	0.1260	1/DAY	1-C		Medvedev & Davidov (1972) •
Mixture of PAH in soils				60% cap.	20 C		5	328	0.0031	1/DAY - R	0	400 (days)	Pace Report (1985)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100% Cap.	20 C		200	1000	0.1000	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100% Cap.	10 C		200	200	0.1020	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils				60% cap.	20 C		50	224	0.1129	1/DAY - R	0	400 (days)	Pace Report (1985)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100% Cap.	30 C		200	730	0.1270	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	123	176	0.3404	mg/kg/day	0-C	575 (days)	RetoC (JH 1987)
Creosote wood preserving waste	FINE SANDY LOAM		6 MANURE		5-30	10 g/100g	167	470	0.3708	mg/kg/day	0-C	575 (days)	RetoC (JH 1987)
Creosote wood preserving waste	McCollum fine sandy loam			80% field cap.	18-22		106.2	99	0.5164	mg/kg/day	0-C	1.0 (days)	RetoC (JH 1987)

DIBENZ(A,H)ANTHRACENE

MEDIA	SOILTYPE	SOILpH	AMNTYPE	MOISTURE	TEMPRANGE	APPLRATE	INITCONC	HALFLIFE	RTECONST	RCUNITS	REACTORSCR	DURATION	STUDYREF
Creosote oil preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	1.38	1.4	2000	0.0003	1/DAY	1	280 days	U.S. EPA (1987)
Creosote wood preserving waste	FINE SANDY LOAM	6	MANURE		5-30	10 g/100g	5	1623	0.0004	1/DAY	1-C	575 (days)	Ref-C (OH 1967)
PCP wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	0.55	2	533	0.0013	1/DAY	1	100 days	U.S. EPA (1987)
Creosote wood preserving waste	FINE SANDY LOAM	6	MANURE		5-30	10 g/100g	5	258	0.0027	1/DAY	1-C	575 (days)	Ref-C (OH 1967)
PCP wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	0.72	1.4	224	0.0031	1/DAY	1	100 days	U.S. EPA (1987)
Pure compound							3.5	190	0.0036	1/DAY	1-C		Sims (1982)
Pure compound							5.7	143	0.0048	1/DAY	1-C		Sims (1982)
Complex fossil fuel waste							27	179	0.0039	1/DAY	1-C		Sims (1986)
Pure compound							4.5	141	0.0049	1/DAY	1-C		Sims (1982)
Pure compound							57	130	0.0053	1/DAY	1-C		Sims (1982)
Pure compound							147	119	0.0058	1/DAY	1-C		Sims (1982)
Pure compound							93	99	0.0070	1/DAY	1-C		Sims (1982)
Complex fossil fuel waste with amendments							27	70	0.0099	1/DAY	1-C		Sims (1986)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100 % Cap.	20 C		10.6	940	0.0056	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100 % Cap.	10 C		10.6	820	0.0076	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)
Mixture of PAH in soils	SANDY LOAM	7.9		80-100 % Cap.	30 C		10.6	750	0.0071	mg/kg/day	0-C	240 (days)	Coover & Sims (1987)

INDENO(1,2,3-cd)PYRENE

MEDIA	SOILTYPE	SOILpH	APPTYPE	MOISTURE	TEMPERATURE	APPLRATE	INITCONC	HALFLIFE	RATECONST	RCUNITS	REACTORCODE	DURATION	STUDYREF
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	1.2%	0.7	330	0.0021	1/DAY	1	280 days	U.S. EPA (1987)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	1.0%	0.6	170	0.0041	1/DAY	1	280 days	U.S. EPA (1987)
Creosote wood preserving waste	Durant Clay Loam			-2 to -4 bars	18-22 C	0.7%	0.6	170	0.0042	1/DAY	1	280 days	U.S. EPA (1987)
Refinery waste	Kidman Sandy Loam	8.1		24.5% (sat.)		25 (MG	9.1	130	0.005	1/DAY	1		A.P.I. (1987)
Creosote wood preserving waste	Durant Clay Loam			-1 bar	18-22 C	1.0%	6.3	230	0.0054	1/DAY	1	100 days	U.S. EPA (1987)
Refinery waste	Kidman Sandy Loam	8.1		24.5% (sat.)		22 (MG	5.6	90	0.007	1/DAY	1		A.P.I. (1987)
Refinery waste	Nunn Clay Loam	8.1		36% (sat.)		25 (MG	5.2	69	0.01	1/DAY	1		A.P.I. (1987)
Refinery waste	Kidman Sandy Loam	8.1		24.5% (sat.)		45 (MG	7.7	63	0.011	1/DAY	1		A.P.I. (1967)
Refinery waste	Nunn Clay Loam	8.1		39% (sat.)		45 (MG	8.5	63	0.011	1/DAY	1		A.P.I. (1987)
API Separator Sludge Waste	Kidman Sandy Loam			-2 to -4 bars	18-22 C	12%	17	62	0.0112	1/DAY	1	280 days	U.S. EPA (1987)
Refinery waste	Nunn Clay Loam	8.1		39% (sat.)		62 (MG,	15.5	58	0.012	1/DAY	1		A.P.I. (1987)

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ATTACHMENT D

EQUATIONS USED TO CALCULATE RISK LEVELS

Model Used to Estimate Target Levels of Carcinogenic Pah in Soil;
Model Incorporates Decay of PAH in Soil

Let:

$C(t)$ = conc in soil at time t (ppm)

$C(0)$ = conc in soil at $t = 0$

t = time measured in years

Shalf = half life of decay in surface soils in years

T = period of exposure in years starting at $t = 0$

$\bar{C}(T)$ = average concentration over exposure interval from $0 < t < T$.

Then:

$$C(t) = C(0)*0.5^{(t/Shalf)}$$

$$C(t) = C(0) * \exp(-(0.693*t)/Shalf)$$

Over interval $0 < t < T$, the average concentration =

$$\bar{C}(T) = C(0)/T * \underset{0 \to T}{\text{Int}} \exp(-(0.693*t)/Shalf)dt$$

After integration and simplification:

$$\bar{C}(T) = (C(0)*Shalf/(T*0.693)) [1- \exp(-0.693*T/Shalf)]$$

$$\bar{C}(T)/C(0) = FRAC = (1/T * Shalf/0.693) [1-\exp(-0.693T/Shalf)]$$

then:

$$C(0) = \bar{C}(T)/FRAC$$

FOR SIMPLE SCENARIOS

A. Exposure Estimate

$$\text{Effective ADD(life)} = \text{dirt} * \text{conc}/1.0\text{E}{+}6 * 1/\text{int} * \text{dur}/\text{life} * \text{matrix}$$

where:

dirt = amt of dirt ingested per event (mg)

conc = concentration of chemical in dirt (ppm)

int = interval between events (e.g.,30 = once every 30 days)

dur = duration over which exposure takes place (yrs)

life = (lifetime of average person = 70 years)

matrix = matrix effect associated with chemical being administered within a soil matrix as compared to the method by which chemical was administered in the experimental tests used to determine toxicity end points. In the present set of calculations the conservative assumption of matrix = 1 has been made.

B. Lifetime Risk Calculation

$$\text{Risk} = (\text{ADD(life)} * \text{Wt. Avg. CPF})/\ \text{body wt}$$

where:

Wt. Avg. CPF was estimated based on the observed relative concentrations of the carcinogenic PAH compounds at the site and the relative potencies of these compounds via the oral intake route. The weighted average potency was estimated to be 1.6661 (mg/kg/day)-1.

Body Wt = average body weight of exposure group

C. Target Level Calculation: Set level equal to 1.0 E-6 (one in one million)

$$\text{Conc} = \frac{(1.0\text{ E}{+}6 * 1.0\text{ E}{-}6) * \text{lnt} * \text{life} * \text{body wt}}{\text{dirt} * \text{dur} * \text{matrix} * \text{Wt. Avg. CPF}}$$

Here Conc is calculated as the average concentration of chemical in soil that would yield an incremental lifetime risk level of one in one million.

Because the concentration of PAH will decrease with time due to biodegradation, we want to calculate the initial concentration in soil C(0) that would result in an average concentration C(T) where this is set equal to Conc. Recall that:

C(0) = C(T)/FRAC

C(0) = Conc/((1/T * Shalf/0.693) [1-exp(-0.693T/Shalf)])

More Complicated Scenarios: 1 through 70 years - unrealistic worst-case scenario

Total lifetime risk = Risk (1-6 yrs) + Risk (6-11 yrs) +
Risk (11-70 yrs)

Perform these operations for 1.0E-6 target risk as follows:

conc = ([(1.0E+6*1.0E-6)*life]/(Wt.Avg. CPF * matrix)) *
([(intA * wtA)/(dirtA*durA)] +(intB * wtB)/(dirtB*durB)] +
[(intC * wtC)/(dirtC*durC)])

where A refers to 1-6 year old period

B refers to 7-11 year old period

C refers to 12-70 year old period

Caveat: for this time varying case, the biodegradation rate should be incorporated mathematically as an integral. The simplification used here introduces a relatively small error.

RETEC
REMEDIATION
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Received 03/24/88

March 23, 1988

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✓ Mr. Tony DeAngelo
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345 Courtland Street
Atlanta, GA 30365

Dear Dr. Walker and Mr. DeAngelo:

At a meeting I had with Dr. Walker on March 15, 1988, Dr. Walker requested that we explicitly state the assumptions used in modeling soil ingestion risks presented in my memorandum titled "Action Levels for the Live Oak Site", dated February 17, 1988. The following assumptions were used in developing the risk factors presented in Table 5 of that memorandum:

Carcinogenic Potency Factor	1.4713 $(mg/kg/day)^{-1}$
Average Soil Concentration at Time 0	55 ppm and 7.6 ppm respectively for the treatment area and the site area based on a composite of the upper two feet of soil.
Matrix Factor for Soil Ingestion	0.3
Frequency of Ingestion	Once every three days
Degradation half-life	0.5 years
Amount of Soil Ingested/Event	100 mg for ages 0-5, 50 mg for ages 6-11 and 5 mg for ages 11-70.

The basis for these assumptions were identified in the memorandum. Dr. Walker requested that we also model the residential exposure risks using the following assumptions:

Amount of Soil Ingested/Event	100 mg for ages 0-11 and 25 mg for ages 11-70.
Degradation Half-Life	0.5 years, 1 year and 1.5 years.

These results are presented in Table 1. Changing the amount of soil ingested from ages 6-70 does not change the results originally presented in Table 5. Increasing the half-life increases the risk only marginally and is still within 10^{-6} for the treatment area and 10^{-7} for the site area. This sensitivity analysis demonstrates that a treatment goal of 100 ppm of carcinogenic PAH is protective of human health and is an appropriate criteria for the site.

It should be noted that the risk levels presented in Table 1 are for unrestricted site development and for surficial soils. Therefore, the requirements in the ROD which pertain to: (a) deed restrictions and (b) soil cover on the treatment area are not necessary components of the final remedy. [illegible]

I trust the above information satisfactorily addresses your question regarding the proposed action levels. Should you require any further clarification please do not hesitate to give me a call.

Sincerely,

John Ryan, ct

John Ryan
Principal

JR:ct

soil cover not necessary
on treat area

10^{-5} [illegible] in

of [illegible]

no land use
restrictions

attempts to
control water
level in land
treat. area

TABLE 1

LIFETIME SOIL INGESTION RISKS ASSOCIATED WITH UNRESTRICTED RESIDENTIAL DEVELOPMENT AFTER TREATMENT OF CONTAMINATED SOILS TO 100 PPM TOTAL PAH CARCINOGENS[a]

HALF LIFE (years)	SOIL INGESTION/EVENT (mg/event)	RISK LEVEL	
		Treatment Area	Site Area[b]
0.5	100 mg for ages 0-5, 50 mg for ages 6-11 and 5 mg for ages 12-70	1.0×10^{-6}	1.4×10^{-7}
0.5	100 mg for ages 0-11, 25 mg for ages 12-70	1.0×10^{-6}	1.4×10^{-7}
1.0	100 mg for ages 0-11, 25 mg for ages 12-70	1.5×10^{-6}	2.1×10^{-7}
1.5	100 mg for ages 0-11, 25 mg for ages 12-70	2.0×10^{-6}	2.8×10^{-7}

---

a) Note: The residential scenario assumes that houses are constructed on the site immediately after treatment is completed and that a person spends his entire life on this site (from birth to 70 years). This scenario is extremely conservative and highly unlikely given the demographics of the area.

b) Weighted average of wood storage area, plant area, treatment area and the overall site.

RETEC
REMEDIATION
TECHNOLOGIES INC

22419 72nd Ave S
Kent WA 98032
(206) 872-0247

Received
03/29/88

March 28, 1988

Dr. Russell Walker
Florida DER
2600 Blair Stone Road
Tallahassee, FL 32399-2400

Dear Russ:

Pursuant to your request, I have calculated the effects of changing the half-life on the risk levels associated with soil ingestion previously presented in my memorandum dated February 17, 1988. Assuming half-life values of 5, 10, and 20 years, the resultant risk levels for unrestricted residential use of the site are:

HALF LIFE (years)	RISK LEVEL	
	TREATMENT AREA	SITE AREA
5	3.7×10^{-6}	5.2×10^{-7}
10	4.2×10^{-6}	6.4×10^{-7}
20	5.2×10^{-6}	7.2×10^{-7}

All other assumptions used in calculating the risk levels are the same as those presented in my memorandum dated February 17, and further clarified in my letter dated March 23, 1988. As we previously discussed, it is ReTeC's position that a half-life of 0.5 years is an appropriate value to use in this evaluation.

This evaluation and the previous data submitted indicate that the risks associated with an action level of 100 ppm carcinogenic PAH for surficial soils is within the 10^{-6} range for the treatment area and 10^{-7} for the overall site as previously reported. It should be noted that these risks are based on the extremely conservative assumption that the site would be used for unrestricted residential development and that such development would occur immediately after treatment of the contaminated soils is completed. The proposed action levels are conservatively low and are protective of human health and the environment.

Please let me know if you have any additional questions.

Best regards,

John Ryan

John Ryan
Principal

JR:ct

cc: T. DeAngelo
K. Burke
K. Paulsen
S. Hugenberg
J. Rodes

BIOREMEDIATION OF CONTAMINATION BY HEAVY ORGANICS AT A WOOD PRESERVING PLANT SITE

By
Ronald J. Linkenheil
Remediation Technologies, Inc.
Fort Collins, Colorado

Thomas J. Patnode
Glacier Park Company
Seattle, Washington

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ABSTRACT

On-site treatment was chosen as the closure alternative for a creosote impoundment at a Superfund site in Minnesota. This alternative was identified in the feasibility study as the most cost effective source control measure for the site. The effectiveness of using land treatment technology to detoxify contaminated soils at the site was demonstrated in pilot scale studies. Results of these studies were used to develop design criteria for a full scale treatment facility.

A lined 3-acre treatment facility was constructed in 1985 treat 10,000 c.y. of contaminated soils and sludges from the creosote impoundment. The facility has been successfully operated by ReTec since 1986 achieving greater than 90 percent reduction of polynuclear aromatic hydrocarbons (PNAs) during the first year of operation. This paper summarizes results from the first year of treatment and demonstrates the effectiveness of the full scale system. Aspects of construction and start-up of the full scale facility are also reviewed.

INTRODUCTION

Wastewaters from a creosote wood preserving operation have been sent to a shallow, unlined surface impoundment for disposal since the 1930's. The discharge of wastewater to the disposal pond generated a sludge which is a listed hazardous waste under the Resource Conservation and Recovery Act (RCRA). Due to groundwater contamination of the shallow aquifer at the site by PNAs, the State of Minnesota nominated the site for listing on the Superfund National Priorities List in 1982. Since 1982 numerous remedial investigation activities have been undertaken to determine the nature and extent of contamination at the site. Based on the results of these studies and extensive negotiations, the Minnesota Pollution Control Agency (MPCA), the U.S. Environmental Protection Agency (EPA), and the owner of the facility signed a Consent Order in March 1985 specifying actions to be taken at the site.

In general terms, the remedial actions selected by the MPCA and EPA involve a combination of off-site control measures and source control measures. The off-site controls involve a series of gradient control wells to capture contaminated ground water. The source control measures include on-site biological treatment of the sludges and contaminated soils and capping of residual contaminants located at depths greater than 5 feet. Costs for on-site treatment and capping were estimated to be $59/ton.

PILOT SCALE STUDIES

Before the on-site treatment alternative was implemented, bench scale and pilot scale studies were conducted to define operating and design parameters for the full scale facility. Several performance, operating, and design parameters were evaluated in the land treatment studies. These included:

- Soil characteristics;
- Climate;
- Treatment supplements;
- Reduction of gross organics and PAH compounds;
- Toxicity reduction;
- Effect of initial loading rate;
- Effect of reapplication;

Three different loading rates were evaluated in the test plot studies: 2 percent, 5 percent, and 10 percent BE hydrocarbons. The soils used in the pilot study consisted of a fine sand which was collected from the upper 2 feet of the RCRA impoundment. The soil was contaminated with creosote constituents consisting primarily of PNA compounds. Total PNAs in the soil ranged from 1000 to 10,000 ppm, and BE hydrocarbons in the contaminated soil ranged from approximately 2 to 10 percent by weight.

Because the natural soils are fine sands and extremely permeable, it was decided that the full scale system would include a liner and leachate collection system to prevent possible leachate break through. To simulate the proposed full scale conditions, the pilot studies consisted of five lined, 50 foot square test plots with leachate collection. The studies were designed to maintain soil conditions which promote the degradation of hydrocarbons. These conditions included:

- Maintain a pH of 6.0 to 7.0 in the soil treatment zone;
- Maintain soil carbon to nitrogen ratios between 50:1 and 25:1; and
- Maintain soil moisture near field capacity.

Hydrocarbon losses in the test plots were measured using benzene as the extraction solvent. The analysis of BE hydrocarbons provides a general parameter which is well suited to wastes containing high molecular weight aromatics such as creosote wastes. Reductions of BE hydrocarbons were fairly similar between all the field plots. Average removals for all field plots over four months were approximately 40% with a corresponding first order kinetic constant (k) of 0.004/day.

The reduction of PNA constituents was monitored by measuring decreases in 16 PNA compounds. The following compounds were monitored in the test plots:

2 Rings

Naphthalene
Acenaphthylene
Acenaphthene

3 Rings

Fluorene
Phenanthrene
Anthracene

4, 5, and 6 Rings

Fluoranthene
Pyrene
Benzo(a)anthracene
Chrysene
Benzo(j)fluoranthene
Benzo(k)fluoranthene
Benzo(a)pyrene
Dibenzo(a,h)anthracene
Benzo(g,h,i)perylene
Indeno(1,2,3,c,d)pyrene

Greater than 62 percent removals of PAHs were achieved in all the test plots and laboratory reactors over a four month period. PAH removals for each ring class are shown below:

- 2 ring PAH: 80-90 percent
- 3 ring PAH: 82-93 percent
- 4+ ring PAH: 21-60 percent
- Total PAH: 62-80 percent

Table 1 summarizes first order rate constants and half-life data for BE hydrocarbons and PNA compounds for the 5 and 10 percent BE hydrocarbon test plots. With the exception of the 4 and 5 ring PNAs, the table shows that the kinetic values are approximately equal for the 5 and 10 percent loading rates. In the case of the 4 and 5-ring compounds, the 5 percent loading rate resulted in higher kinetic rates for these compounds as compared to the 10 percent

TABLE 1
COMPARISON OF PILOT SCALE KINETIC DATA AT TWO INITIAL LOADING RATES

	First Order Rate Constant (day^{-1})		Half-life (days)	
	5 % Plot	10 % Plot	5 % Plot	10 % Plot
Benzene Extractable	0.003	0.003	231	231
2-Ring PAH	0.023	0.023	30	30
3-Ring PAH	0.016	0.016	43	43
4-Ring PAH	0.004	0.001	173	693
TOTAL PNAs	0.009	0.008	77	87

loading rate. This difference may have been due to more 2-ring and 3-ring compounds being available to soil bacteria at the 10 percent loading rate. These compounds may be preferentially degraded by soil bacteria.

OPERATING AND DESIGN CRITERIA

The pilot scale studies were successful in developing operating and design criteria for a full scale system. These criteria are summarized below:

- Treatment period can be extended through October
- Soil moisture should be maintained near field capacity
- Soil pH should be maintained between 6.0 and 7.0
- Soil carbon:nitrogen ratios should be maintained between 25:1 and 50:1
- Fertilizer applications should be completed in small frequent doses
- Initial benzene extractable hydrocarbon contents of 5 to 10% are feasible
- Waste reapplication should occur after initial soil concentrations have been effectively degraded
- Waste reapplication rates of 2 to 3 lb of benzene extractable per cubic foot of soil per 3 degradation months can be effectively degraded

The studies suggest that all the loading rates tested are feasible. First order rate constants were fairly similar between all the test plots although the intermediate loading rate (5% benzene extractable hydrocarbons) may demonstrate a slightly higher removal of high molecular weight PAH compounds. The higher loading rates, however, showed the greatest mass removals. The selection of an initial loading rate should balance additional land area requirements against time requirements for completing the treatment process. Moderate loading rates (5%) will result in a faster detoxification whereas higher loading rates will decrease land a requirements.

CONSTRUCTION AND START-UP OF FULL SCALE SYSTEM

Construction of the full scale system involved preparation of a treatment area within the confines of the existing RCRA impoundment (Figure 1). The treatment area was constructed on top of the impoundment to avoid permitting a new RCRA facility. If the facility was located outside the impoundment, then a Part B permit would have to be obtained before the treatment facility could be constructed. By locating the treatment area within the confines of the impoundment, the treatment system was considered part of closure of the impoundment. This enabled us to fast track the clean-up and avoid the delays associated with permitting a new RCR unit.

The principal construction activities at the site involved:

- Preparation of a lined waste pile for temporary storage of the sludge and contaminated soil.
- Removal of all standing water in the impoundment.
- Excavation and segregation of the sludges for subsequent free oil recovery.
- Excavation of approximately 3-5 feet of "visibly" contaminated soil from the impoundment and subsequent storage in the lined waste pile.
- Stabilization of the bottom of the impoundment as a base for the treatment area.
- Construction of the treatment area including installation of a 100 ml HDPE liner, a leachate collection system and 4 feet of clean backfill.

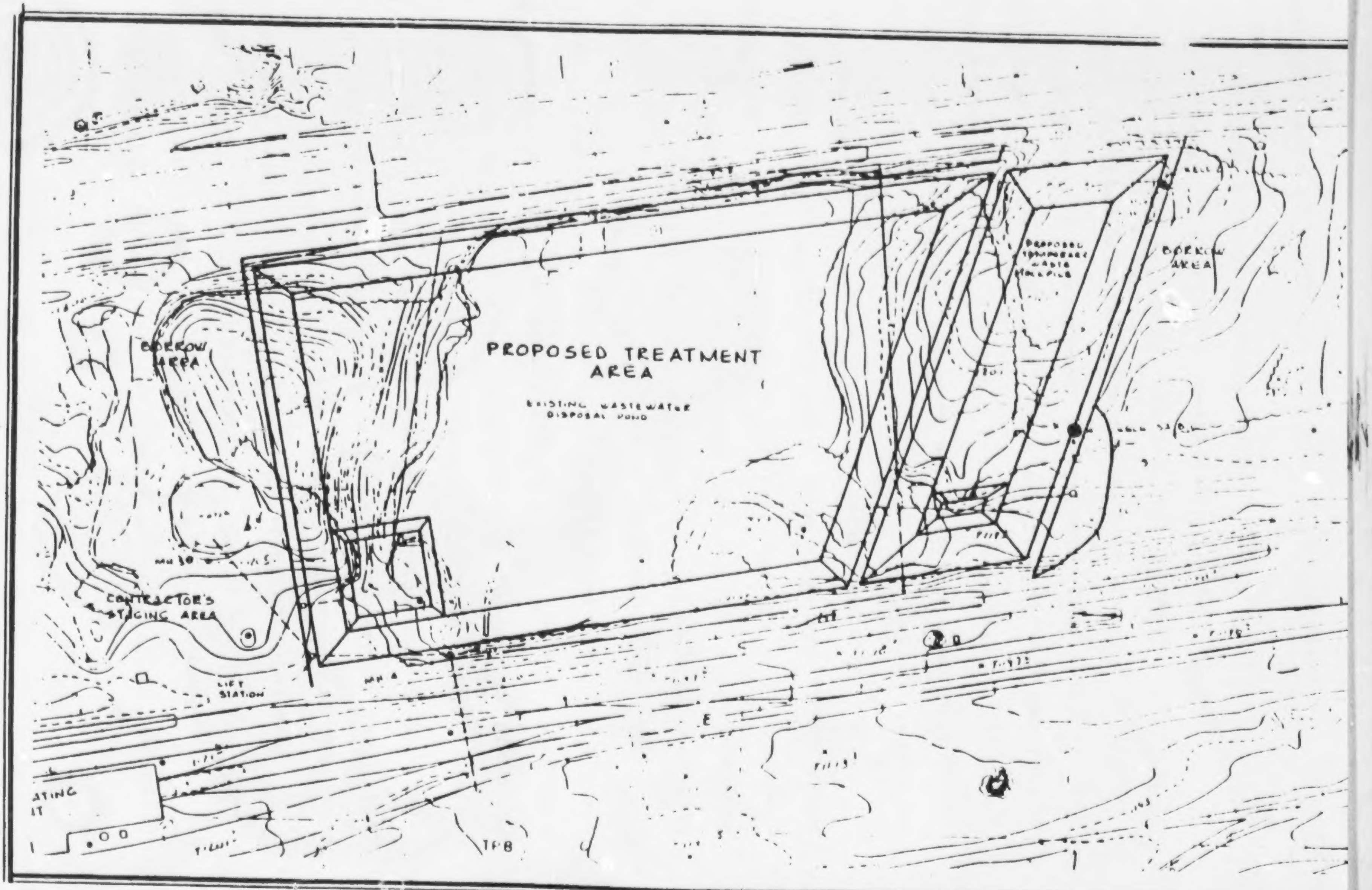

PROPOSED TREATMENT AREA
EXISTING WASTEWATER DISPOSAL POND
BORROW AREA
BORROW AREA
PROPOSED
CONTRACTOR'S STAGING AREA
LIFT STATION
MH 30
MH A
TP8

Figure 1. Site Plan for On-site Treatment System

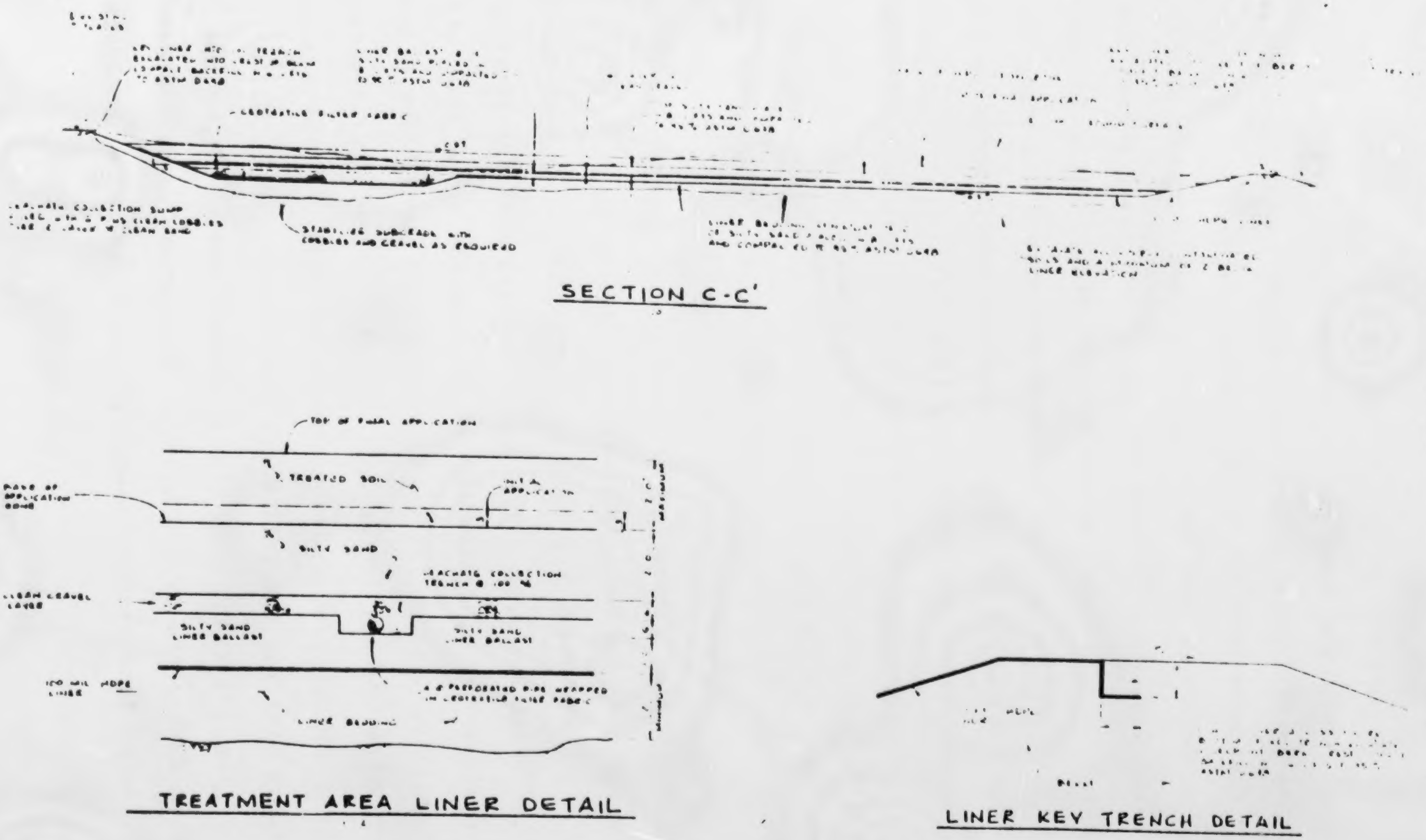
- Installation of a sump for collection of the stormwater and leachate.
- Installation of a center pivot irrigation system.

As previously discussed, a lined treatment area w constructed because the natural soils at the site are highly permeable. A cap also was needed for the residual contaminants left in place below the liner. Therefore, the treatment area liner serves two functions at the site. The first function is to provide a barrier to leachate from the treatment area. The second function is to provide a cap over the residual contaminants that were left in place.

The treatment area was constructed on top of the existing waste water disposal pond after all contaminated materials were removed. The surface area for treatment is approximately 125,000 square feet. Containment berms with 3 to 1 slopes enclose the treatment area and prevent surface run off from leaving the site.

The treatment area is lined with a 100 mil HDPE membrane (Figure 2). The base of the liner slopes 0.5 percent to the south and west. A sump with a 50,000 gallon capacity is located in the southwest corner of the treatment area. A layer of silty sand ballast 18 inches thick was placed on top of the treatment area liner. A 6 inch gravel layer was placed on top of the ballast. This layer serves as a leachate collection system and as a marking layer for land treatment operations.

The leachate collection system includes 2 foot wide leachate collection drains at 100 foot centers (Figure 2). The drains are filled with gravel and perforated pipe to carry leachate from the collection system to the sump. The drains were wrapped in filter


GEOTEXTILE FILTER FABRIC
STABILIZE SUBGRADE WITH COBBLES AND GRAVEL AS REQUIRED
SECTION C-C'
TOP OF FINAL APPLICATION
TREATED SOIL
INITIAL APPLICATION
SILTY SAND
LEACHATE COLLECTION TRENCH @ 100 %
CLEAN GRAVEL LAYER
SILTY SAND LINER BALLAST
100 MIL HDPE LINER
PERFORATED PIPE WRAPPED IN GEOTEXTILE FILTER FABRIC
LINER BEDDING
TREATMENT AREA LINER DETAIL
LINER KEY TRENCH DETAIL

Reproduced from best available copy.

Figure 2. Cross Section and Liner Details for Treatment Area

fabric to prevent clogging. Two feet of uncontaminated sand was placed above the leachate collection system. This layer of sand serves as an initial mixing layer for the contaminated soils and the treatment zone for the full scale system.

Water in the leachate collection sump is discharged by gravity flow to a manhole and is automatically pumped via a lift station to a 117,000 gallon storage tank. Water in the storage tank is recycled back to the treatment area via a spray irrigation system. Water in excess of irrigation requirements is discharged to the municipal wastewater treatment plant.

Construction of the waste pile and treatment area was completed in October 1985. In late April 1986, a center pivot irrigation system was installed and 120 tons of manure were spread in the treatment area. Manure loading rates were based on achieving a carbon:nitrogen ratio of 50:1. In addition to nitrogen, the manure provides organic matter which enhances absorption of the hazardous waste constituents.

In May 1986, a 3 inch lift of contaminated soil was applied to the treatment area. The target loading rate for start-up was a BE hydrocarbon concentration of 5 percent. The soil was mixed (rototilled) with 3 inches of native soil to achieve a treatment depth of 6 inches. This application involved approximately 1200 cubic yards of sludge and contaminated soil. Table 2 summarizes start-up data for the full scale facility.

The treatment area is irrigated almost daily due to dry weather during the summer months. Irrigation needs are determined from soil tensiometer readings, soil moisture analyses, and

TABLE 2
SUMMARY OF START-UP DATA (5/23/86)

Parameter	Average
Benzene Extractables, %	53000
TOC, ppm	29710
TKN, ppm	1367
Ammonia, ppm	2.37
Total Phosphorus, ppm	522
Total Potassium, ppm	502
pH	7.66
Polynuclear Aromatic Hydrocarbons (PAH), ppm:	
Naphthalene	1148
Acenaphthylene	21
Acenaphthene	1082
Total 2-Ring PAH	2251
Fluorene	1885
Phenanthrene	4190
Anthracene	3483
Total 3-Ring PAH	9558
Fluoranthene ✓	1575
Pyrene	958
Benzo(a)anthracene and Chrysene	837
Total 4-Ring PAH	3370
Benzofluoranthenes	368
Benzopyrenes	294
Indeno(123cd)pyrene	111
Dibenzo(ah)anthracene	100
Benzo(ghi)perylene	106
Total 5-Ring PAH's	979
Total PAH's	16159

precipitation and evaporation records. Typical irrigation rates range from 1/4 inch to 3/8 inch per application. This application rate keeps the soils in the cultivation zone moist witho saturating soils in the lower treatment zone. Maintaining soil moisture near field capacity was determined to be a key operating parameter in the pilot scale studies.

PERFORMANCE OF THE FULL SCALE FACILITY

Benzene extractable (BE) hydrocarbons and 16 polynuclear aromatic (PNA) compounds are being monitored to evaluate the performance of the facility. Figure 3 shows the BE hydrocarbon concentrations measured in the Zone of Incorporation (ZOI) during the first year of treatment. BE hydrocarbon concentrations decreased approximately 60 percent over the first year of operation. Most of the decrease occurred during the first 120 days (May through September). Little decrease in BE hydrocarb concentrations was observed during the Fall and Winter months.

Figures 4 and 5 show PNA concentrations measured in the treatment facility during the first year of treatment. Figure 4 summarizes data for 2-ring and 3-ring PNAs. Figure 5 summarizes data for the 4-ring and 5-ring compounds. Greater than 95 percent reductions in concentration were obtained for the 2 and 3 ring PNAs. Greater than 70 percent of the 4-ring and 5-ring PNA compounds were degraded during the first year of operation.

With the exception of anthracene, all the 2-ring and 3-ring compounds were degraded below or near detection limits after 90 days of treatment. Greater than 92 percent of the anthracene

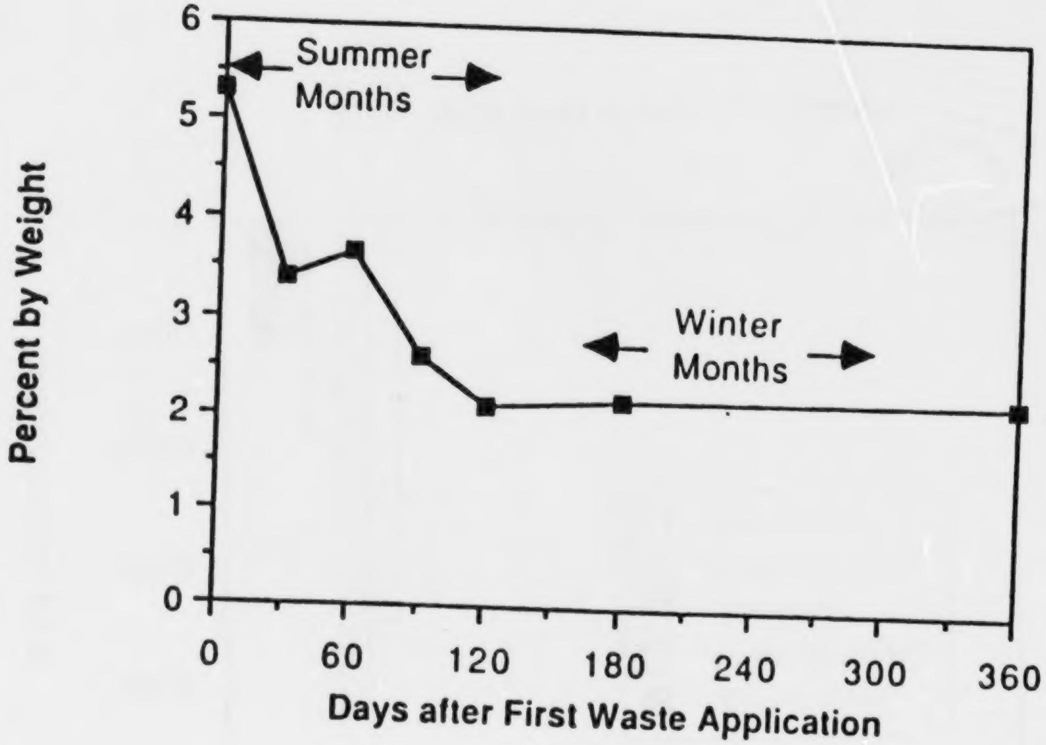


Figure 3. BE Hydrocarbons Degradation vs Time

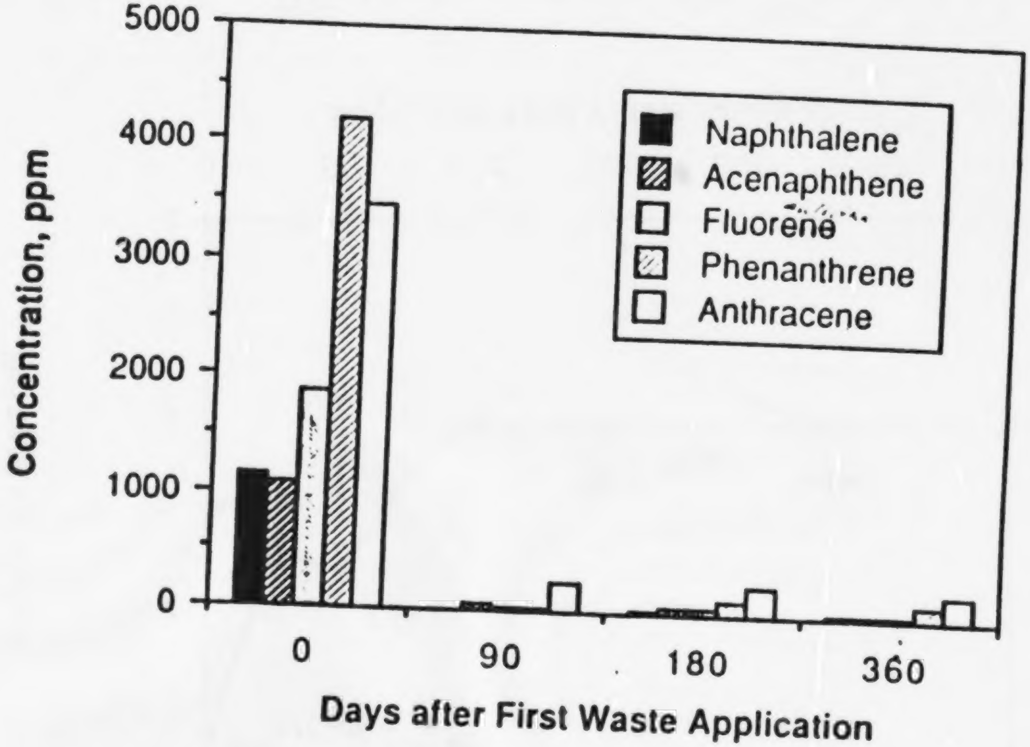


Figure 4. 2-Ring and 3-Ring PNA Degradation vs Time

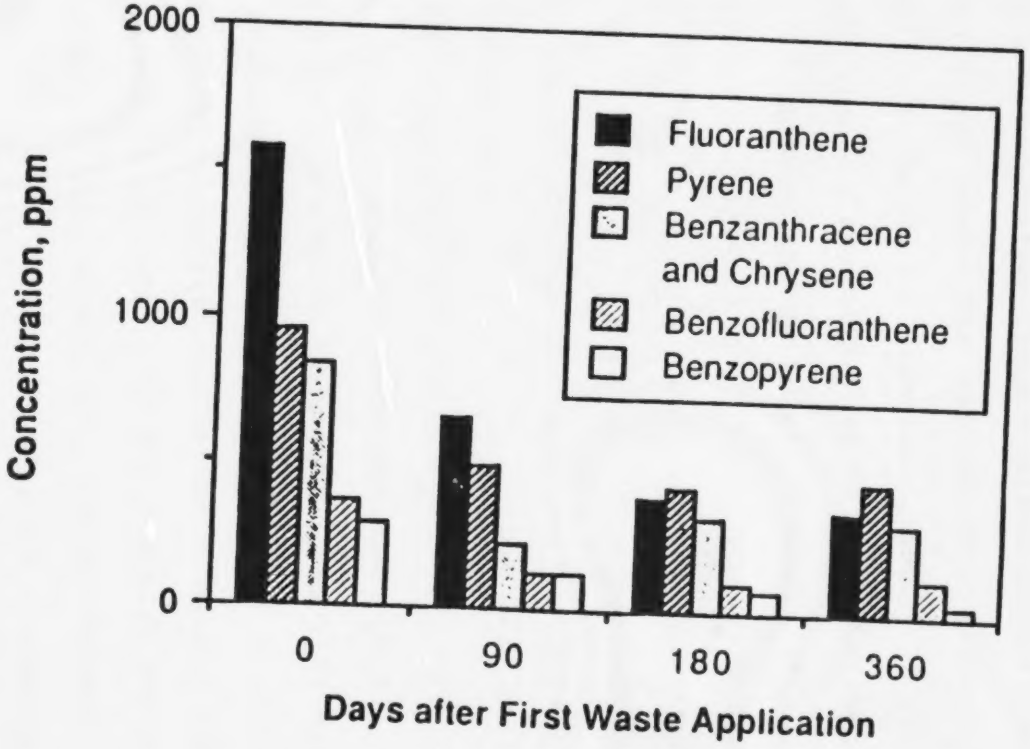


Figure 5. 4-Ring and 5-Ring PNA Degradation vs Time

present in the waste was degraded during the first 90 days of treatment. Similarly, most of the 4 and 5 ring removals occurred during the first 90 days of treatment. This was expected becau the warmest weather occurred during the this period.

Table 3 shows average PNA removals measured in the pilot scale studies and compares them with the full scale removal efficiencies. Full scale removal efficiencies were higher than test plot removal efficiencies for every PNA ring class and BE hydrocarbons. However, it must be noted that the full scale facility operated for 360 days compared to only 126 days for the test plot units. Table 3 also presents average half-life data for both the test plots and the full scale unit. Full scale half-lives were consistently in the low end of the range of half-lives reported for the test plot units.

In summary, the rate and amount of PNA degradation is proportional to the number of rings contained by the PNA compounds (Figure 6). The 2-ring and 3-ring PNAs degraded most rapidly. The 4-ring and 5-ring PNAs degraded at slower rates, however, these compounds are strongly adsorbed to soils and are immobilized in the treatment zone of the facility. Table 4 summarizes water quality data for the leachate collection system of the facility. Only acenaphthene and fluoranthene were detected in the drain tile water samples. Concentrations for these two compounds were near analytical detection limits.

CONCLUSION

The data developed during this project has shown that on-

TABLE 3
COMPARISON OF FULL SCALE AND TEST PLOT REMOVALS

Parameter	AVE. PERCENT REMOVAL		AVE. HALF-LIFE (DAYS)	
	Full Scale[1]	Test Plots[2]	Full Scale	Test Plots
2-Ring PAHs	95	93 - 95	<45	29 - 33
3-Ring PAHs	95	83 - 85	45	46 - 49
4- and 5-Ring PAHs	72	32 - 60	115	95 - 226
Total PAHs	90	65 - 76	65	61 - 83
BE Hydrocarbons	60	35 - 56	150	106 - 202

[1] Removal efficiency calculated after 193 days of treatment.

[2] Removal efficiency calculated after 126 days of treatment.

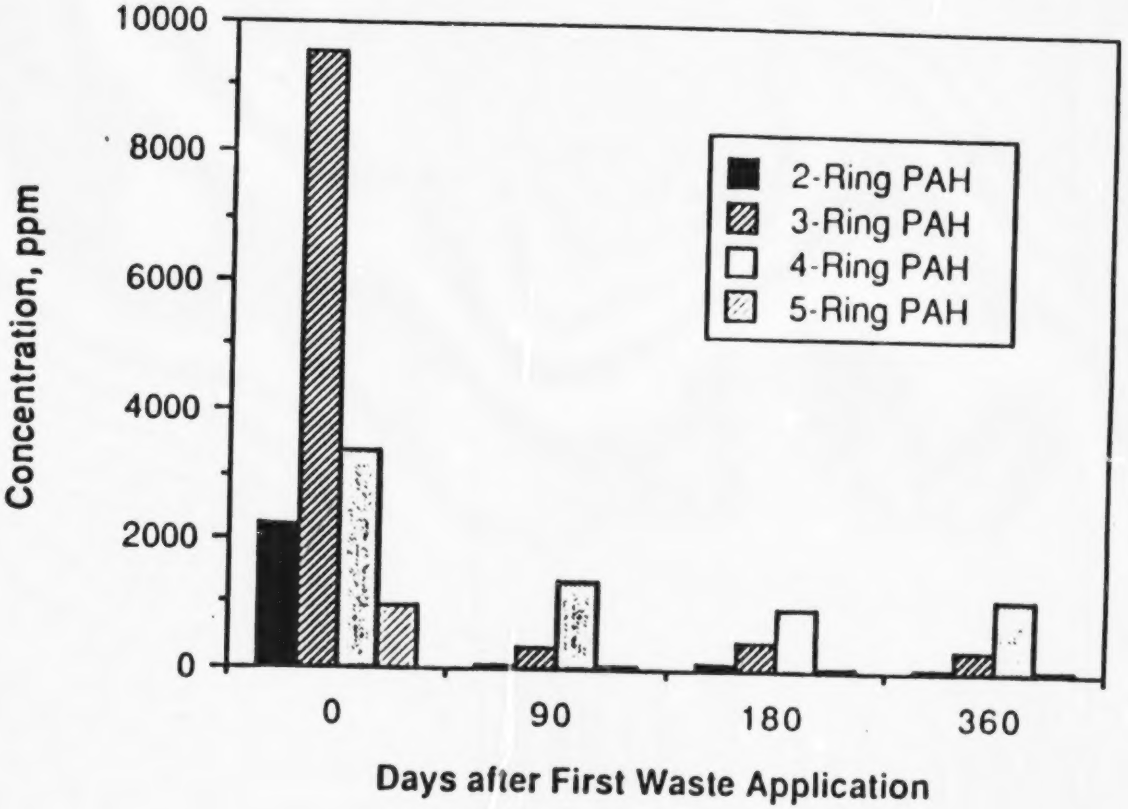


Figure 6. PNA Degradation by Ring Class

TABLE 4
DRAIN TILE WATER QUALITY

Compound	Concentration, ppb		
	June 1986	August 1986	October 1986
Naphthalene	<1	<1	<1
1-Methylnaphthalene	<1	<1	<1
2-Methylnaphthalene	<1	<1	<1
Acenaphthylene	<1	<1	<1
Acenaphthene	<1	3.7	2.7
Fluorene	<1	<1	<1
Phenanthrene	<1	<1	<1
Anthracene	<1	<1	<1
Fluoranthene	<1	2.1	1.4
Pyrene	<1	<1	<1
Benzo(a)anthracene	<1	<1	<1
Chrysene	<1	<1	<1
Benzofluoranthenes	<5	<1	<1
Benzopyrenes	<5	<1	<1
Indeno(123cd)pyrene	<5	<1	<1
Dibenzo(ah)anthracene	<5	<1	<1
Benzo(ghi)perylene	<5	<1	<1

site treatment of creosote contaminated soils is feasible. Based on the data developed in pilot scale studies, a conservative design for a full scale system was developed and constructed. The full scale unit has matched or surpassed the performanace of the pilot scale unit in degrading creosote organics. The advantages of on-site treatment are that it reduces the source of contaminants at the site in a very cost effective manner. In addition, it satisfies the developing philosophical approach that EPA has to on-site remedies and it reduces the liability of the owner/operator due to off-site disposal.

ATTACHMENT C

State Letter of Concurrence

STATE OF FLORIDA
DEPARTMENT OF ENVIRONMENTAL REGULATION

TWIN TOWERS OFFICE BUILDING
2600 BLAIR STONE ROAD
TALLAHASSEE, FLORIDA 32399-2400

BOB MARTINEZ
GOVERNOR

DALE TWACHTMANN
SECRETARY

April 6, 1988

Mr. Greer Tidwell
Regional Administrator
U.S. Environmental Protection
 Agency, Region IV
345 Courtland Street, NE
Atlanta, Georgia 30365

Dear Mr. Tidwell:

The Florida Department of Environmental Regulation concurs with the selection of Alternative F. for remediation of the Brown Wood Preserving Superfund site in Live Oak, Florida, as described in Section II.F. of the Record of Decision.

This alternative consists of off-site disposal of highly contaminated soil at an EPA-approved hazardous waste facility, and on-site treatment of mildly contaminated soil by biodegradation. All soil having greater than 100 mg/kg of total carcinogenic indicator compounds is to be disposed of or treated. If biodegradation of contaminated soil fails to attain the cleanup goal of 100 mg/kg total carcinogenic indicator compounds after two years of treatment, then an alternate remedy will be determined by EPA at that time.

The remedy selected provides an effective means of reducing the long term threat to public health and the environment posed by the site. Upon conclusion of a five-year groundwater monitoring program, no further operation or maintenance will be necessary. This remedy will be implemented by two potentially responsible parties, AMAX, Inc. and the James Graham Brown Foundation.

We look forward to the successful completion of this remedial action.

Sincerely,

Dale Twachtmann

Dale Twachtmann
Secretary

DT/aet

ATTACHMENT D

Letter from DOI re Natural Resources Survey

United States Department of the Interior

OFFICE OF THE SECRETARY
WASHINGTON, D.C. 20240

4

ER-84/552

JUL 20 1984

Mr. Gene Lucero, Director
Office of Waste Programs Enforcement
Environmental Protection Agency
Washington, D.C. 20460

Dear Mr. Lucero:

Pursuant to your request we have conducted a preliminary natural resources survey at the Brown Wood Preserving site, Live Oak, Suwannee County, Florida, to determine whether any natural resources under the trusteeship of the Secretary of the Interior have been, are being, or have the potential to be affected by releases of hazardous substances at the site.

Our survey revealed that there are no lands, minerals, waters, or Indian resources under Interior's trusteeship in the vicinity of the site. Although certain fish and wildlife, including endangered species, under our trusteeship inhabit the vicinity, there is no evidence of damages to these resources at the site itself. Moreover, the probability of off-site damages is remote.

Accordingly we would be willing to grant a release from claims for damages to natural resources under the trusteeship of the Secretary of the Interior in regard to releases of hazardous substances at the Brown Wood Preserving site.

Sincerely,

Bruce Blanchard, Director
Environmental Project Review

cc: Janet Farella

ATTACHMENT E

Transcription re the Public Meeting on October 9, 1987

Minutes of the Public Meeting

Date: December 09, 1987

Location: Live Oak City Hall, Live Oak, Florida

Time: 2 to 5 PM

Subject: The Proposed Remedy for the Brown Wood Preserving National Priorities List Site, Live Oak, Suwannee County, Florida

Note: Transcribed from cassette tape by Tony DeAngelo, 12/16/87

DeAngelo: This is the public meeting for the Brown Wood Superfund Site which is located over on Sawmill and Goldkist. If you have not signed in, please do so so that we have a reading on who attended the meeting.

First, I'd like to introduce the regulatory people here. My name is Tony DeAngelo. I'm an engineer and the Superfund Project Manager and work out of the EPA Regional office in Atlanta. This gentleman (points to his right) is Mike Henderson. He works for the Office of Congressional and External Affairs at the Regional office in Atlanta. He's our PR man. This gentleman here (points to his left) is Charles Rooks. He's the attorney who works out of the Regional office and is assigned to this case.

This gentleman is John Ryan from ReTech. He is a consultant for AMAX and the Brown Foundation.

Also, we have Cindy Hilty, a Supervisor from the FDER in Tallahassee. Russ Walker, PhD, who's the State Project Manager who works on the Site. The other gentleman here I don't know. If he would introduce himself,....

Joe Rodes: I'm Joe Rodes.

DeAngelo: The purpose of this meeting is to present our proposed remedy for the site and to solicit any comments from anybody and how our proposed remedy might be modified. If anyone here has not been down to the library, I encourage you to go down to library and at least browse through the documents down there. Under Section 113 of the Law, the Superfund Law, there's what we call an administrative record placed in that repository down there. From time to time new documents will be sent down to the repository, ...

Angelo: At this time I would like to present a brief description of the Superfund process so that you can get a general idea of how the Superfund process works and where we are in the process. The initial step in the process is the location and identification and grading of a large number of sites nationwide as regards their potential or probability to cause hazard or to be hazardous to the public health, welfare and the environment. We have a system whereby we do preliminary assessments and site investigations and state grants are given for that purpose. Out of this large number of sites, about over 20,000 at this time in the nation, we cull those which have a very high priority. These are subjected to the Hazard Ranking System scoring, a score between 0 and 100. It goes through a very detailed verification process in Washington; and from there the sites are verified to have that hazard and are placed on the National Priority List. Brown Wood is one of those sites that was placed on the List. At this time there are about 1,000 sites nationwide on the National Priority List. Brown Wood ranks approximately in the middle or perhaps a little below middle on the List. From this large number of sites that we get from culling down, we get those sites that we focus a lot of attention on....

They are treated in a very special manner. There's a process whereby we do what we call a remedial investigation/feasibility study which is essentially a study which defines the nature and extent of the contamination and then comes up with alternatives for cleanup. From ther we go to what we call a decision document or the record of decision. At that time EPA's comments and the public's comments and the potentially responsible parties' comments are concentrated and we come up with a remedy which is approved by EPA. From there we go into what we call the remedial design/remedial action stage where we design the actual remedy and then the remedial action carries it out. After that the site is once again subjected to an investigation, mainly of files, and we come up with a deletion report. That is submitted to Headquarters and then finally, hopefully, the site is deleted from the National Priority List and is deemed to be safe. Where Brown Wood is at the moment is: we have a remedial investigation/feasibility study done. And a record-of-decision hopefully will be signed by the end of this month (December) and at that time we will begin negotiating with the potentially responsible parties, AMAX and the Brown Foundation and any other potentially responsible parties we can bring to bear; and see if they want to implement the remedial design/ remedial action. You might ask why is this present removal activity going on out there.........

being undertaken under Section 122 (e)(6) of the Superfund Law which indicates that this type of activity can go on if authorized by the President. The activity is in concert with the present proposed draft remedy and isn't deemed to be hazardous or causing any kind of endangerment to the public. Unless anyone has any questions

DeAngelo: about this, what I've said already, I'll..................

of course most of the people here understand the basic history of
the site. It was a wood preserving ....
It basically operated for 30 years from 1948 through 1978. The
preservatives used were creosote and pentachlorophenol, the
acronym for that is PCP. There are several areas where the
creosote and the PCP have contaminated the surface soils ; and
there is a lagoon out there which has creosote sludges in it.
As far as we know at this time there's no significant groundwater
contamination. Chiefly due to the probable existence of a layer
of clay underneath the sludge in the lagoon and also because
there are a great many channels under the site which allow the
groundwater to move rather rapidly. Since the last release over
10 years ago.........................most of the contamination
....
EPA's proposed remedy for the site consists of several parts.
Number 1: we propose that the sludges in the lagoon and any
other sludges that we find be removed and pretreated or treated
and be taken to a facility at Emelle, Alabama, or Pinewood,
South Carolina. Two: we propose that a test demonstration using
one or two cells using contaminated surface soil............
....

....
Three: we also propose that

Four: ......

I believe that pretty well describes what the EPA proposes; so
so I'd like to open it up ....to questions or comments.
(No questions or comments forthcoming.)
Okay.
I think that the present removal action is probably very appropriate
at this time. There are several reasons for that. Frankly, we at
the Regional office have received some flak for authorizing this
activity. However, there are overlaps of window of opportunity
that we can take advantage of at the present time, the chief one is
the dry season in this part of the South. It will cost a great deal
more if we operate in the wet season and it will present greater
problems...........
Number two, there is a difficult regulatory situation at the present
time. Under the Resource Conservation and Recovery Act, and
Amendments to that Law, or additions to that, there is a gradual
Land Ban going into effect whereby only certain wastes can be taken
from these sites and sent to hazardous waste facilities. It's a
gradual phase-in. There have been variances given so that certain
wastes go in at certain time periods. This type of waste here,
creosote wastes, ...can go in only for a
very short period of time. When that window closes, given the

present situation, then it can no longer be done. I was told by Headquarters within the last couple of days that virtually all this type of waste will eventually have to be incinerated on-site or off-site. The type of land disposal whereby excavation and removal was accomplished will be a thing of the past........

Are there any questions? (Pause) Any questions?

Cindy Hilty: With regard to the Land Ban, what does it say exactly? RCRA facilities will no longer be allowed to accept creosote wastes ....What does it exactly cover?

Tony DeAngelo:
I gave ....documents to Russ (Walker)

What you will find is that it becomes difficult to know what to do with the wastes given the lead times that you have. It's going to become difficult to plan ahead given these varying windows of opportunity.

Chuck Rooks: Let me just clarify that: when Tony said that there was some flak on conducting the removal operation. Nobody is questioning the environmental safety or anything like that. It was simply a matter of how it was handled at the Regional office and it doesn't present any kind of environmental harm.............. we think that the is appropriate and everyone agrees with that.

Dennis Price(FDER local liaison):

Tony DeAngelo:
Does anyone have any questions about the enforcement process? Exactly how things are going to operate after the record-of-decision is signed.

Someone: I'm sure we do.

Tony DeAngelo:
Well, all right, I want to say a few words about that. There are two sides to the Superfund process in terms of not only immediate removals, chemical spills and that kind of stuff, but also in terms of remedial or long-term activity at sites like Brown Wood. The one side is what we call the Fund lead side. In that area EPA people act in concert with EPA contractors to accomplish such activities. On the other side we have the enforcement area. That is where EPA technical people and legal personnel get together and meet with people we feel are potentially responsible under the Law for what's occurred at the site and try to work out agreements

to accomplish cleanups............................ It's generally a more laborious process.
In the case of Brown Wood, if we had not done anything, not authorized any activity within the last couple of months, in other words, if that removal had not been done, then it would probably be at least one whole year before anyone would be out on the site again doing any kind of cleanup work. Under the new law we have to go through a consent decree process. A consent decree is an agreement between the potentially responsible parties and EPA wherein the potentially responsible parties agree to do certain work within certain time frames. That Decree is passed on by a judge in a Federal District Court. The Decree is not presented by a Regional attorney, but by the Department of Justice. So you can see that the mechanisms are set up so that it's probably going to take quite a bit longer in order to come to an agreement to actually do the actual cleanup.

I would appreciate any questions ——
I'm willing to take a shot......
(Unintelligible.)

Cindy Hilty: (She says something about looking forward to seeing something from Retech.)
(Unintelligible.)

Tony DeAngelo:
Do you have any? Okay, if there's no other questions we will stay around awhile to answer any questions you might have. I appreciate y'all coming and I look forward to seeing you in the future. Thankyo